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VOL. I.—16TH YEAR. SYDNEY, SATURDAY, JUNE 15, 1929. No. 24.

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Table of Contents

THE OCCURRENCE OF LEAD IN THE EGG OF THE DOMESTIC HEN.[1]

PART III.

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(Submitted for Publication, April 19, 1929.)

INTRODUCTION.

THE results presented here are the outcome of a suggestion that heavy elements should be sought in eggs of the domestic hen. This material was employed by the Bio-physical Section of the Cancer Research Committee for certain X ray experiments and it was thought that the presence of heavy elements would explain some of the results obtained. Previous papers[10][11] have given the results obtained from a preliminary survey of egg material for lead and also a detailed account of the method employed in its estimation.

It has been stated by Needham[29] that the only inorganic elements, other than those commonly accepted as present, occurring in eggs are arsenic,[6] boron,[7][8] copper,[16] fluorine,[10][33] iodine,[12][18] manganese,[9] sulphur[23] and iron.[20] Aluminium has recently been reported by Myers and others[27] to be present and denied by McCollum, Rask and Becker.[24]

Lead has been reported as being normal to man's internal organs by Barse,[4] Chevallier,[13] Devergie[15] and Orfila.[31] General results since these workers tend to show that lead is not a natural constituent of the body, but it may be found in healthy persons who have been quite free from symptoms of lead poisoning. So far as can be determined, lead has not been previously reported as occurring in eggs and of its absorption, as well as of the other elements given above, by embryonal tissue during growth nothing is known.

The chemistry and physiology of the developing avian egg has been reported by Liebermann[23] and in numerous papers by Murray[26] and Needham;[28] little of the work reported by these authors has been repeated here and then only when of interest to the work in hand.

Many attempts to isolate and identify the lead compound in tissues from animals with induced lead poisoning have been reported principally by Fairhall[3][17] and Minot,[25] leading to their conclusion that lead circulates in the blood stream most probably as colloidal lead phosphate[3] and not as the albuminate.[30] Experiments reported here indicate that the lead is present in egg yolk as a fat-soluble compound, as outlined in an earlier paper.[11]

SCOPE OF WORK.

In this paper are presented results showing:

1. The weights of eggs from the same hen over a period of seven months.
2. The loss in weight of the egg during incubation.
3. The weights of embryos from the same hen's egg at different ages.
4. The absorption of lead during incubation by the embryo.
5. The injection of different lead compounds into the egg, their absorption and toxicity.
6. The nature of the lead compound in egg yolk.

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

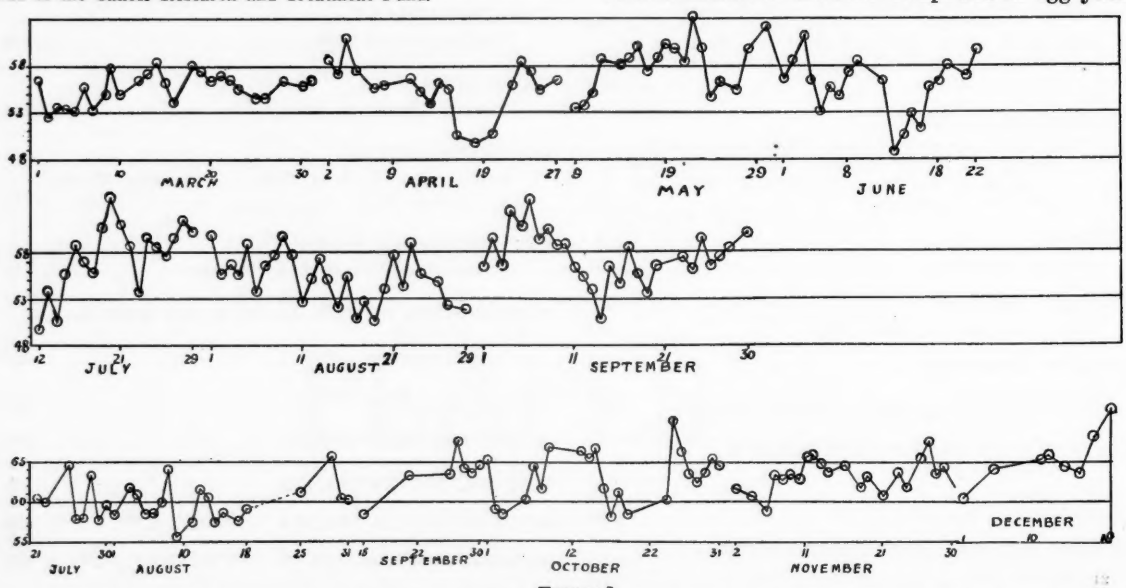


FIGURE I.
Showing weight in grammes of eggs from the same hen. The upper chart from white Leghorns and the lower from black Orpingtons.

TABLE I.

Black Orpington Hen Eggs.

Date.	Weight in Grammes.	Date.	Weight in Grammes.	Date.	Weight in Grammes.
1927. July		1927. September		1927. November	
21	60·3	15	58·5	2	61·8
22	60·0	21	63·2	4	60·7
25	64·4	26	63·2	6	58·9
26	57·7	27	67·5	7	63·1
27	58·0	28	64·0	8	62·9
28	63·4	29	63·4	9	63·4
29	57·5	30	64·5	10	62·8
30	59·4			11	65·4
		October		12	65·8
		1	65·1	13	64·9
		2	59·0	14	63·7
August		3	58·5	16	64·5
1	58·2	6	60·1	18	61·7
3	61·9	7	64·7	19	63·1
4	60·8	8	61·5	20	60·9
5	58·5	9	66·9	22	63·7
6	58·5	13	66·2	23	61·8
7	60·0	14	65·4	25	65·4
8	64·0	15	66·5	26	67·7
9	55·7	16	61·5	27	63·3
11	57·3	17	58·1	28	64·2
12	61·7	18	61·1	30	60·7
13	60·3	19	58·4	December	
14	57·3	24	60·3	1	60·4
15	58·5	25	70·6	5	64·1
17	57·3	26	66·0	11	65·1
18	59·0	27	63·5	12	65·9
25	61·1	28	62·4	14	64·5
29	65·6	29	63·7	16	63·5
30	59·2	30	65·5	18	68·1
31	59·2	31	64·4	19	71·9

White Leghorn Hen Eggs.

Date.	Weight in Grammes.	Date.	Weight in Grammes.	Date.	Weight in Grammes.
1928. March		1928. May		1928. July	
1	56·5	9	53·5	17	57·0
2	52·8	10	53·9	18	55·9
3	53·9	11	55·0	19	60·7
5	53·2	12	58·7	20	63·8
6	56·0	14	58·2	21	60·9
7	53·4	15	58·9	22	58·7
8	54·7	16	60·1	23	53·7
9	57·5	17	57·6	24	59·4
10	55·0	18	58·4	25	58·5
12	56·5	19	60·2	26	57·6
13	57·3	20	59·9	27	59·7
14	58·4	21	58·4	28	61·4
15	56·2	22	63·2	29	60·2
16	54·0	23	60·0	August	
18	58·0	24	54·7	1	59·8
19	57·6	25	56·3	2	55·6
20	56·5	27	55·4	3	56·8
21	57·0	28	59·8	4	55·4
22	56·6	30	62·2	5	58·7
23	55·9	June		6	53·9
25	54·8	1	56·8	7	56·5
26	54·7	2	58·9	8	57·4
28	56·5	3	60·6	9	59·9
30	56·0	4	56·5	10	57·6
31	56·8	5	53·0	11	52·4
April		6	55·8	12	55·3
2	58·7	7	54·7	13	57·3
3	57·2	8	57·2	14	54·2
4	60·9	9	58·7	15	52·0
5	57·8	12	56·5	16	55·4
7	55·9	13	48·8	17	50·9
8	56·1	14	50·1	18	52·7
11	56·8	15	52·8	19	50·5
12	55·2	16	51·2	20	54·0
13	54·0	17	55·7	21	57·8
14	56·5	18	56·5	22	54·0
15	54·7	19	58·2	23	58·9
16	50·5	21	57·0	24	55·6
18	49·9	22	59·6	26	54·9
20	50·8	July		27	51·2
22	56·1	12	49·6	29	50·9
23	58·6	13	53·7	September	
24	57·4	14	50·4	1	56·4
25	55·5	15	55·6	2	59·4
27	56·7	16	58·8	3	56·5

TABLE I (*Continued*).

White Leghorn Hen Eggs (Continued).

Date.	Weight in Grammes.	Date.	Weight in Grammes.	Date.	Weight in Grammes.
1928. September		1928. September		1928. September	
4	62·4	12	55·4	20	56·7
5	60·6	13	53·9	23	57·4
6	63·5	14	50·6	24	56·0
7	59·2	15	56·4	25	59·5
8	60·2	16	54·6	26	56·4
9	58·8	17	58·5	27	57·6
10	58·7	18	55·6	28	58·2
11	56·2	19	53·6	30	60·0

THE WEIGHT OF EGGS FROM THE SAME HEN.

Cook[14] has determined the weights of eggs in one dozen lots and states that the average weight per egg is 53·4 grammes. It was noticed that the eggs from the white Leghorn hens were smaller than those from black Orpington hens. The weights of eggs from one fowl of each breeding were tabulated, when it was noted that after a period of rest the weight of the eggs was always low, rising rapidly to a more normal value. Generally, before cessation of laying there would be a very light egg some six to eight days before the rest period, the last egg being of average weight. This is shown in Table I and in Figure I.

LOSS IN WEIGHT DURING INCUBATION.

The eggs were incubated and weighed from day to day, the temperature being 38·2° C. ± 1·8° C. and the humidity as given by the wet and dry bulb thermometer 68%. The reason for the above temperature variation was that the door of the incubator was opened several times daily for other purposes. When the incubator was placed in a room of almost constant temperature the above variations were lessened. Forty-four eggs were weighed every day and the weight plotted against age as shown in Figure II; the loss in weight per day is given in Table II and graphically in Figure

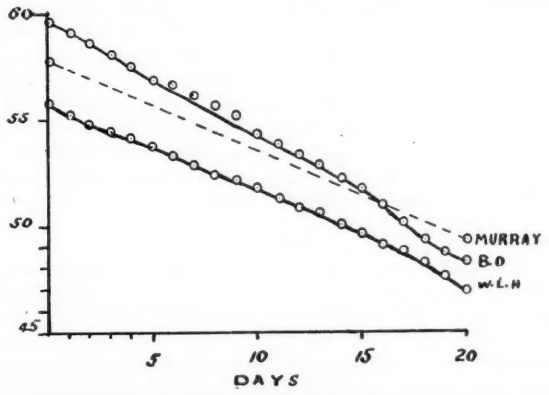


FIGURE II.

Loss of weight of eggs during incubation. The average weights of twenty-two white Leghorn (W.L.H.) and twenty-two black Orpington (B.O.H.) hen eggs with the mean value obtained by Murray for white Leghorn hen eggs. Abscissa represents weight in grammes.

TABLE II.

Showing Loss of Weight in Grammes of Incubated Eggs. (Temperature = 38·2° ±1·8° C. Humidity = 68%.)

Variety.	No. of Eggs.	Average Weight.	Average Loss per Day.	Loss of Weight per Day.																	
				1st Day.	2nd Day.	3rd Day.	4th Day.	5th Day.	6th Day.	7th Day.	8th Day.	9th Day.	10th Day.	11th Day.	12th Day.	13th Day.	14th Day.	15th Day.	16th Day.	17th Day.	18th Day.	19th Day.	20th Day.
White Leghorn	22	55·70	0·438	0·4	0·5	0·35	0·32	0·49	0·40	0·42	0·47	0·30	0·30	0·45	0·40	0·40	0·50	0·45	0·44	0·39	0·47	0·62	0·63
Black Orpington	22	59·58	0·573	0·38	0·55	0·56	0·56	0·60	0·41	0·52	0·48	0·50	0·65	0·50	0·45	0·60	0·65	0·70	0·78	0·90	0·80	0·60	0·40

III. For comparison a graph of Murray's figures for similar determinations on white Leghorn eggs is also given.[26]

It is shown by the graphs that the average weights of the Leghorn eggs are less than those of Murray's and may be due to the age of the hens which in the above series was about one year. In Figure III the variations in the three sets of eggs are well shown. Only those for the Leghorn eggs can be compared with Murray's figures.

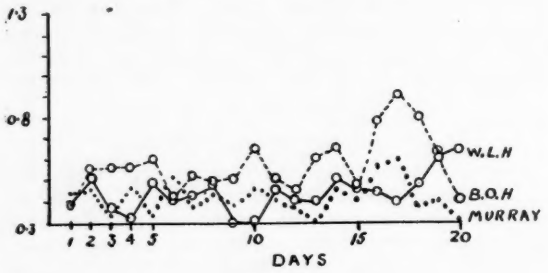


FIGURE III.

Loss in grammes per day of eggs during incubation, white Leghorn (W.L.H.) and black Orpington (B.O.H.) hen eggs. Murray's mean values for white Leghorn hens are shown for purposes of comparison.

Weight of Embryo During Incubation.

The weights of the developing avian embryo have been given in detailed studies of Murray[26] and Needham.[28] The results of Needham do not agree with extrapolated values of Murray, although the embryos were derived from the same white Leghorn hens. It was therefore decided to repeat these observations on eggs from two fowls of different breeding, namely one black Orpington and one white Leghorn hen, the eggs of each being kept separate, so forming two series and also avoiding individual differences due to different fowls.

Technique.

The incubation was conducted under conditions as near those given by Murray[26] as it was possible to attain. The temperature was 38·2° C. ± 1·8° C. with a humidity of 60% to 70%. Ventilation was effected by a continuous circulation of warm air, the eggs being aired every morning for fifteen to twenty minutes according to room temperature and rolled once each day.

It was essential to have a clean separation of embryo from the remainder of the egg. The method suggested by Needham[28] was found most satisfactory. Before opening the egg, it was placed over an electric "spot-light" and the area occupied by white was marked in pencil on the shell. The shell was then cracked inside this area and the white allowed to escape through a gradually enlarged opening. The yolk was allowed to escape in a similar manner. The embryo was thus left intact in the shell. It was then tipped into a flat shallow dish, illuminated from below, freed from adhering membranes, allowed to drain free of liquid. Any remaining liquid was removed by a capillary tube, to the end of which a rubber teat was attached. The embryo was then weighed in a tared closed weighing bottle.

Fertile eggs of from five to twenty days' incubation were opened as described. With the very early embryos errors due to loss of blood and other sources were never minimal; only with the older embryos was there any certainty that errors from these sources were least. The results given in Tables III and IV thus represent the mean average weights of embryos from white Leghorn and black Orpington eggs, varying from six to twenty days, incubated at 38·2° C. ± 1·8° C. and 60% to 70% humidity.

Curves of the average weights are given in Figure IV with Murray's and Needham's figures for comparison.

TABLE III

Weights of Embryos from White Leghorn Fowls kept at Baulkham Hills, Incubated under Conditions given above.

Age in Days	Number Weighed.	Wet Weights (Grammes).			Dry Weights (Grammes).		
		Bishop.	Murray.	Needham.	Bishop.	Murray.	Needham.
6	50	0·604	0·423	0·322	0·0302	0·0236	0·0176
7	31	0·969	0·735	0·579	0·0555	0·0430	0·0333
8	30	1·613	1·189	0·962	0·1030	0·0738	0·0584
9	4	2·435	1·817	1·503	0·1750	0·1181	0·0959
10	5	3·406	2·661	2·239	0·2662	0·1863	0·1522
11	4	4·549	3·750	3·205	0·3704	0·2888	0·2375
12	2	5·692	5·105	4·427	0·602	0·4495	0·3691
13	2	6·792	6·839	5·472	0·753	0·6905	0·570
14	4	7·842	8·974	7·906	1·072	1·099	0·895
15	1	10·563	11·46	10·21	1·663	1·674	1·386
16	1	13·051	14·39	12·92	2·201	2·360	2·017
17	1	16·142	17·950	16·17	3·091	3·090	2·725
18	3	20·050	22·03	19·99	3·250	3·887	3·488
19	2	24·175	26·670	24·35	5·475	4·719	4·303
20	1	27·95	—	29·83	7·370	—	5·159

Murray[26]. Needham[28].

TABLE IV.

Weights of Embryos from Black Orpington Fowls kept at Dulwich Hill.

Age in Days.	Number Weighed.	Weights in Grammes.	
		Wet.	Dry.
6	50	0·949	0·049
7	50	1·026	0·061
8	50	2·013	0·133
9	20	3·052	0·212
10	20	4·106	0·306
11	10	5·243	0·443
12	10	6·154	0·654
13	4	7·324	0·844
14	2	7·992	1·092
15	1	11·041	1·751
16	1	15·220	2·620
17	1	18·546	3·296
18	1	23·453	4·203
19	1	27·321	6·021
20	1	30·507	7·807

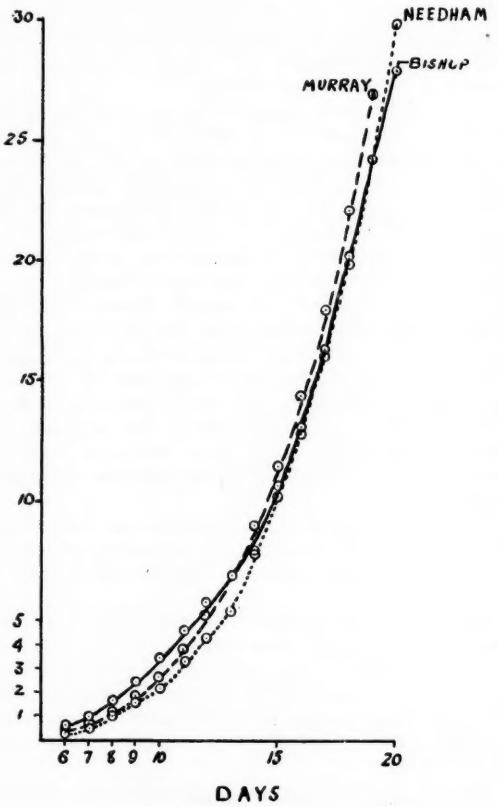


FIGURE IV.

Showing the average wet weight in grammes of chicken embryos as a function of their age, illustrating Table III.

Ash in Embryos.

Previous determinations of the ash content of embryos have been recorded by Liebermann,[23] Murray[26] and Needham.[28] None of these workers has stated what is meant by the term "ash," so it is impossible to compare results obtained by the author with those previously recorded. In the following estimations "ash" means the hydrochloric acid extract of the carbonized ash. The chlorides were weighed after drying at 100° C. in a steam oven. In Table V the results of ash determinations of white Leghorn embryos are given and also Murray's figures, although they cannot be compared, because the nature of the ash is not stated. Table VI presents a similar series of determinations with black Orpington embryos.

TABLE V.

The Ash Weighed as Chloride, Obtained from White Leghorn Embryos.

Age in Days.	Dry Weight.	Ash in Grammes.	Ash. %	Murray. Ash. %
6	0·0302	0·0059	19·5	14·3
7	0·0555	0·0187	19·4	13·8
8	0·1030	0·0194	18·8	13·3
9	0·1750	0·0315	18·0	12·8
10	0·2660	0·0426	16·0	12·2
11	0·370	0·0563	15·2	11·6
12	0·602	0·0845	14·0	10·8
13	0·753	0·100	13·3	9·8
14	1·072	0·1298	12·1	8·7
15	1·663	0·1899	11·4	7·9
16	2·201	0·2201	10·0	7·5
17	3·091	0·2660	8·6	7·5
18	3·250	0·3350	10·3	7·7
19	5·475	0·575	10·5	8·0
20	7·370	0·825	11·2	8·4

TABLE VI.

The Ash Weighed as Chlorides, Obtained from Black Orpington Embryos.

Age in Days.	Dry Weight, Grammes.	Ash, Grammes.	Ash, %
6	0·049	0·0100	20·4
7	0·062	0·0123	19·75
8	0·133	0·0253	19·0
9	0·212	0·0397	18·7
10	0·306	0·0536	17·5
11	0·443	0·0762	17·2
12	0·654	0·1051	16·1
13	0·844	0·1268	15·0
14	1·092	0·1488	13·6
15	1·751	0·2195	12·5
16	2·620	0·2882	11·0
17	3·2960	0·3496	10·5
18	4·203	0·4510	10·7
19	6·021	0·6623	11·0
20	7·807	0·8820	11·3

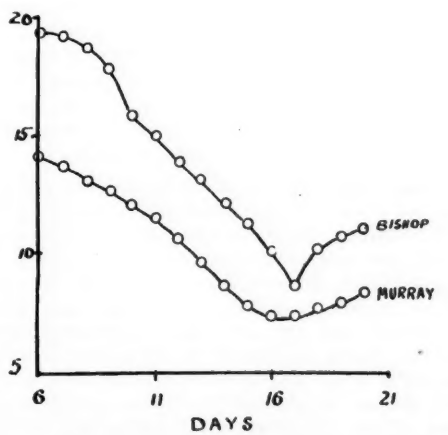


FIGURE V.

Showing the percentage ash (as hydrochloride) calculated on the dry weights of embryos. The graph for Murray's results expresses the theoretical values deduced from an equation given by that author.

A point of interest shown in both Table V and Table VI is that the fall in ash percentage ceases on the seventeenth day, when there is a slight rise until hatching takes place.

THE ABSORPTION OF LEAD BY THE EMBRYO DURING DEVELOPMENT.

As has been shown in an earlier paper[11] lead has been found in all hens' eggs examined. It was also shown that the variation in the lead content of the eggs lay between fairly large limits which were least when the eggs from any one fowl were considered. Hence in the experiments which follow, as well as in those already recorded, the embryos were obtained from the eggs of one fowl, each series thus being a record of the lead absorbed by the embryo during growth from eggs from the one fowl, thus reducing variations in lead to a minimum. For the very early embryos (to the seventh day) embryos were collected from three fowls of the same breed, after a preliminary examination had shown that the lead value was not appreciably affected. These fowls were kept under the same yard and food conditions as the fowl whose eggs subsequently completed the series.

The Estimation of Lead.

For the estimation of lead in the embryos and egg material the Nessler method was used, a critical account of which has appeared elsewhere.[10]

The method adopted for the estimation of lead in eggs and chicken embryos is as follows: The organic material is dried in a large hot air oven at 105° C. until a dry mass capable of being pulverized is obtained. This condition was never attained with egg yolks owing to the high content of natural fat. The dry material is ashed in a silica basin standing on quartz pebbles within a similar basin, the whole being surrounded by an iron flue and heated by a small Bunsen burner, until a hard graphitic residue is obtained. By this means the material is ashed evenly, there being no excessive heating at the bottom of the basin which never exceeds a barely visible red heat. The hard ash, moistened with distilled water to prevent loss by dust, is ground in the silica basin with an agate pestle to a fine powder. The end of the pestle is rinsed with distilled water into the dish.

To the powdered ash are added ten cubic centimetres of concentrated hydrochloric acid (purified by distillation) for each one hundred grammes of original wet material and the basin placed on a hot water bath for two hours. During this period distilled water is added to replace that lost by evaporation and the ash also thoroughly stirred. After two hours the liquid in the basin is decanted through a filter paper into a glass evaporating basin; the ash is washed five times, ten cubic centimetres of hot distilled water being used for each wash and the filtrates placed in a glass evaporating basin and evaporated to dryness.

To the ash in the basin are added a further ten cubic centimetres of hydrochloric acid and the ash is heated for one hour on the hot water bath. The liquid is decanted through the filter into the same glass basin as before and the ash washed free of chlorides with hot distilled water. The combined filtrates are evaporated to dryness.

The residue is dissolved in water, made just acid to methyl orange with hydrochloric acid, diluted to twenty cubic centimetres, one drop of copper acetate solution (2%) is added and sulphuretted hydrogen passed into the solution for one quarter of an hour. This period is sufficient to precipitate all the lead present. The test tube is then stoppered and allowed to stand for twenty-four hours. The precipitate is rinsed into Pyrex glass centrifuge tubes (fifteen cubic centimetres capacity) with sulphuretted hydrogen water and spun for fifteen minutes at three thousand to four thousand revolutions per minute. The clear supernatant liquid is syphoned off and the precipitate washed with three cubic centimetres of sulphuretted hydrogen water and again centrifuged for fifteen minutes. This process is repeated four times.

The precipitate is dissolved in the tubes by adding one drop of concentrated nitric acid and warming on the water bath. Evaporate nearly to dryness (when completely dry the precipitate is difficult to redissolve) and add one cubic centimetre of pure concentrated sulphuric acid (Merck's "pro analysi"). The tube is heated to strong fuming, cooled and four cubic centimetres of distilled water-alcohol mixture (3:1) are added. Stand over night.

The tubes are centrifuged for fifteen minutes, carrying down the lead sulphate. The precipitate after the top liquid has been syphoned off is washed three times with five cubic centimetres of the following mixture: Alcohol (98%) 32 volumes, concentrated sulphuric acid three volumes, water 65 volumes. Centrifuge for fifteen minutes after each wash.

The precipitate of lead sulphate in the tubes is then dissolved in five cubic centimetres of hot 1:2 ammonium acetate solution, cooled and Nesslerized as follows:

The solution is placed in a ten cubic centimetre graduated cylinder, one cubic centimetre of 10% potassium cyanide solution is added, one cubic centimetre of 0·880 ammonium hydroxide and two drops of colourless sodium sulphide solution are added and the whole is made up to ten cubic centimetres with distilled water.

The comparison is then made with the following solution: Five cubic centimetres of a 1:2 ammonium acetate solution, one cubic centimetre of a 10% potassium cyanide solution, one cubic centimetre of 0·880 ammonium hydroxide, two drops of sodium sulphide and the whole is made up to nine cubic centimetres with distilled water. This solution must be quite colourless. Sufficient of the standard lead acetate solution is then added from a burette, until the colour developed is the same as that of the unknown solution. The amount of lead standard solution added is read off from the burette, which should be graduated to read 0·05 cubic centimetres.

The solutions are then placed in the cups of a Duboscq colorimeter and the final exact comparison is made.

The standard lead solution is made from lead acetate and should contain approximately 0·1 milligramme of lead per cubic centimetre. This solution should be freshly prepared, as the value alters rapidly, as has been already noted elsewhere, unless at least 1·0% of free glacial acetic acid is added.

The following additional points should be noted.

The method of ashing is important, as the whole of the material is thus as nearly as possible at the same low red heat, obviating local heating. Lead has an appreciable vapour pressure at temperatures above 600° C. and it is essential to char the organic material below this temperature. Owing to the ready access of air which does not hold when the char is placed in a muffle, the time of ashing is considerably shortened.

Lead only should be present in the final stages of comparison. This is achieved by washing the lead sulphate thoroughly with the acid-alcohol-water mixture. Any traces of copper remaining are prevented from interfering in the reaction by the addition of potassium cyanide.

For the preparation and examination of the reagents, the original paper should be consulted.

The Absorption of Lead.

The results are given in Table VII and Table VIII. The increase in lead per day of embryo

TABLE VII.

Eggs from Fowl at Baulkham Hills, having an Average Lead Content of 0·035 milligramme for Combined Yolk and White per Egg. Embryos obtained under conditions given previously.

Column 1. Age in Days.	Column 2. Number of Embryos used for Analysis.	Column 3. Weights in Grammes of Embryos.		Column 4. Milligramme of Lead Found		Column 5. Milligrammes of Lead per 100 Grammes of Embryo.		Column 6. Lead Absorbed Each Day as Percentage of what remains to be absorbed.		Column 7. Column 6 calculated to 100 Grammes of Embryo Material.		
		Wet.	Dry.	In Embryo.	In Yolk-White.	Wet.	Dry.	Day	%	Day.	Wet.	Dry.
6	50	0·604	0·0392	0·0003	0·035	0·0497	0·994	—	—	—	—	—
7	31	0·969	0·0555	0·0006	0·035	0·0620	1·08	6–7	0·857	6–7	109·0	2,001
8	30	1·613	0·1030	0·0009	0·035	0·0559	0·874	7–8	0·857	7–8	66·4	1,081
9	4	2·435	0·175	0·001	0·034	0·0412	0·571	8–9	0·290	8–9	14·34	208·5
10	5	3·406	0·266	0·0012	0·034	0·0353	0·451	9–10	0·588	9–10	20·1	267
11	4	4·549	0·370	0·0021	0·0345	0·0463	0·567	10–11	2·62	10–11	66·0	825
12	2	5·692	0·602	0·003	0·035	0·0528	0·498	11–12	2·59	11–12	50·5	533
13	2	6·792	0·753	0·0045	0·035	0·0664	0·599	12–13	4·29	12–13	68·7	633
14	4	7·842	1·072	0·0060	0·034	0·0765	0·561	13–14	4·35	13–14	59·5	476
15	1	10·563	1·663	0·009	0·0345	0·0853	0·541	14–15	8·71	14–15	94·6	638
16	1	13·051	2·201	0·012	0·034	0·0921	0·545	15–16	8·71	15–16	73·7	451
17	1	16·142	3·091	0·021	0·024	0·130	0·680	16–17	31·0	16–17	212·1	1,170
18	3	20·05	3·25	0·024	0·014	0·119	0·740	17–18	15·7	17–18	86·9	496
19	2	24·175	5·475	0·027	0·010	0·112	0·493	18–19	25·0	18–19	113	580
20	1	27·950	7·370	0·035	0·005	0·125	0·475	19–20	168·0	19–20	645	2,619

TABLE VIII.

Eggs from Black Orpington Fowl at Dulwich Hill, having an Average Lead Content of 0·089 milligramme for Combined Yolk and White per Egg. Embryos obtained under conditions given previously.

Column 1 Age in Days.	Column 2. Number of Embryos used for Analysis.	Column 3. Weights in Grammes of Embryos.		Column 4. Milligramme of Lead Found		Column 5. Milligrammes of Lead per 100 Grammes of Embryo.		Column 6. Lead Absorbed Each Day as Percentage of what remains to be absorbed.		Column 7. Column 6 calculated to 100 Grammes of Embryo Material.		
		Wet.	Dry.	In Embryo.	In Yolk-White.	Wet.	Dry.	Day.	%	Day.	Wet.	Dry.
6	50	0·949	0·049	0·0008	0·080	0·0844	1·63	—	—	—	—	—
7	50	1·026	0·061	0·0010	0·085	0·0975	1·64	6–7	0·236	6–7	23·9	430
8	50	2·013	0·128	0·0015	0·080	0·0746	1·17	7–8	0·625	7–8	41·2	662
9	20	3·052	0·212	0·0024	0·085	0·0785	1·13	8–9	1·06	8–9	41·9	624
10	20	4·106	0·306	0·0036	0·085	0·0875	1·17	9–10	1·41	9–10	39·5	348
11	10	5·243	0·543	0·0050	0·080	0·0955	0·92	10–11	1·70	10–11	36·4	402
12	10	6·154	0·654	0·0080	0·080	0·130	1·225	11–12	3·75	11–12	66·0	626
13	4	7·324	0·854	0·010	0·075	0·1365	1·17	12–13	2·58	12–13	41·9	342
14	2	7·992	1·092	0·017	0·070	0·213	1·55	13–14	9·55	13–14	124·9	984
15	1	11·041	1·771	0·025	0·065	0·226	1·41	14–15	11·82	14–15	124	825
16	1	15·220	2·620	0·035	0·055	0·230	1·335	15–16	16·70	15–16	127	763
17	1	18·546	3·296	0·050	0·035	0·269	1·520	16–17	33·40	16–17	197	1,131
18	1	23·453	4·203	0·065	0·020	0·276	1·540	17–18	54·50	17–18	260	1,452
19	1	27·321	5·221	0·075	0·020	0·275	1·440	18–19	50·0	18–19	205·8	1,060
20	1	30·507	7·807	0·080	0·005	0·262	1·020	19–20	40·0	19–20	140·5	614

growth is given in column 4 and graphically in Figure VI, which is a regularly ascending curve without points of interest.

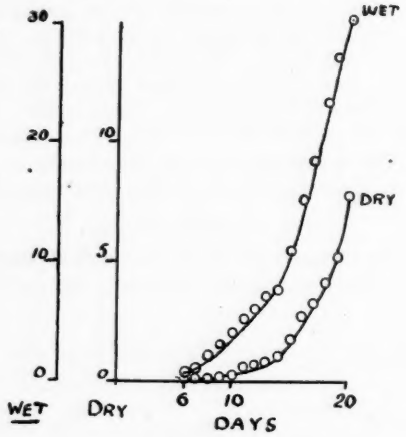


FIGURE VI.
Showing curve for Column 3 of Table VII. Abscissæ represent weight in grammes.

In Figure VII we have column 5 graphed. The wet weight curve, after a rise to the seventh day followed by a depression to the tenth, shows a continuous rise to the seventeenth day, falling away to the nineteenth and then rising again to the twentieth. The dry weight curve shows the same

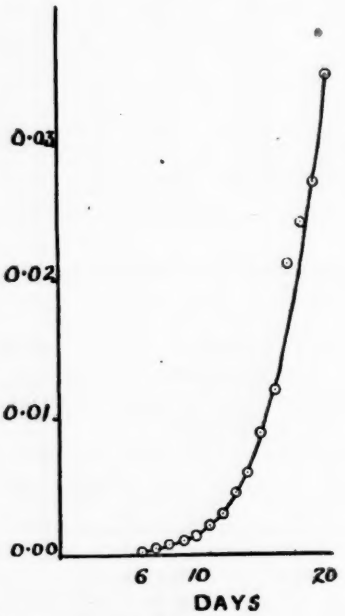


FIGURE VII.
Showing curve for Column 4 of Table VII. Abscissa represents weight in milligrammes.

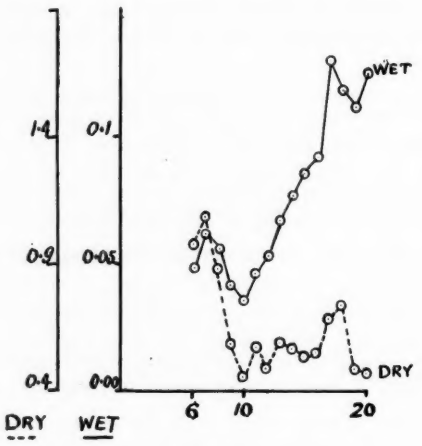


FIGURE VIII.
Showing curve for Column 5 of Table VII. Abscissæ represent weight in milligrammes.

peak and the same depression, but as the embryo is growing much drier as it increases in size the curve falls away more rapidly.

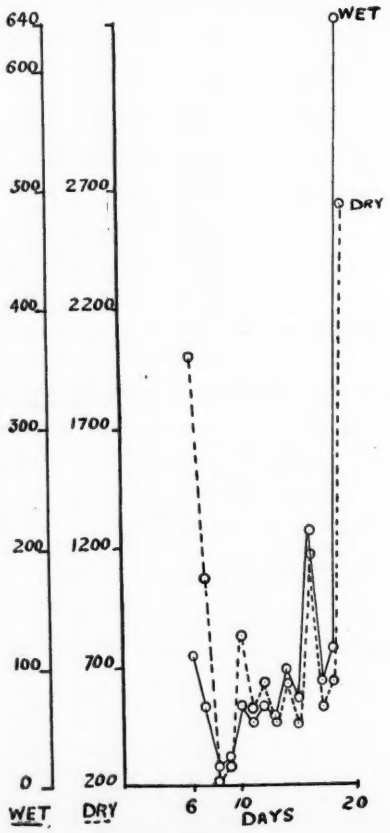


FIGURE IX.
Showing Curve for Column 7 of Table VII.

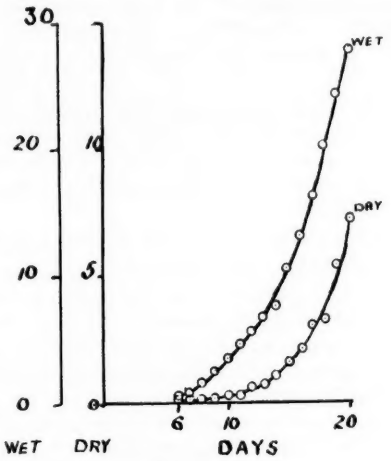


FIGURE X.

Showing curve for Column 3 of Table VIII.

In column 6 is given the absorption of lead as milligrammes absorbed from day X to day Y expressed as percentages of what remains to be absorbed at that time. These values correspond to what one hundred milligrammes of lead in the egg (exclusive of the shell) give to the embryo between any two days of its development. This curve rises similarly to the growth curves and gives little information unless related to a constant weight of embryonal tissue. Hence the absorption from day X to day Y *per centum* of what remains to be absorbed was therefore expressed in percentages of the wet and dry weights of the embryos. These figures represent what one hundred milligrammes of

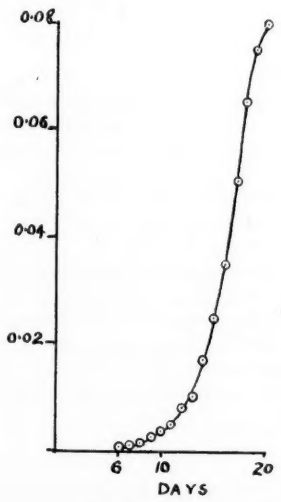


FIGURE XI.

Showing curve for Column 4 of Table VIII.

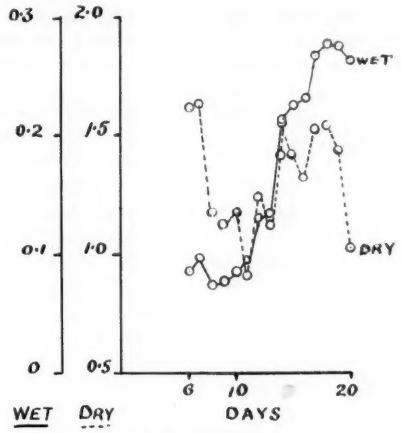


FIGURE XII.

Showing curve for Column 5 of Table VIII.

lead in the egg give to one hundred grammes of wet (or dry) weight of embryo between any two days of its growth and are given graphically in Figure VIII and Figure XI.

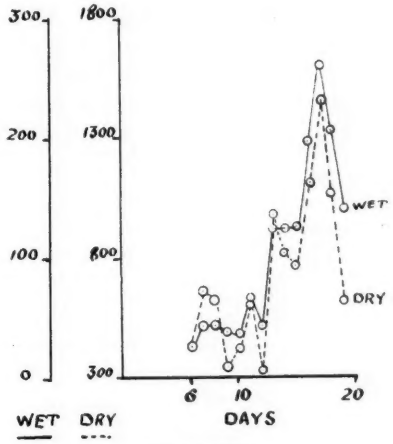


FIGURE XIII.

Showing Curve for Column 7 of Table VIII.

INJECTION EXPERIMENTS.

The injection experiments recorded here were performed in the hope that some indication might be gained from them as to the possible nature of the lead compound in the eggs. It is obvious, since the lead is absorbed by the developing embryo, that the lead must be combined in some form innocuous to the normal development of the embryo. Thus it was thought that the injection of various lead salts into eggs subsequently incubated, would show those compounds which were least toxic to the embryo, and also those most readily absorbed.

The toxicity of lead compounds to embryonal cells has been examined in detail by Bell.[5] It is evident, since the chicken embryo absorbs lead from the yolk

during development, that it has what may be termed a definite "lead toleration" and that concentrations of lead below a definite value do not apparently adversely affect the embryo.

Preliminary experiments showed that the toxicity of lead was entirely dependent on the lead compound used. Thus of injections with lead nitrate and lead oleate the lead content of each injection being the same, that with lead nitrate prevented germination, while with the lead oleate development was normal, although the hatched chicken was weakly and did not live more than two days. Chickens hatched from eggs injected with lead compounds were always found to be very much physically impaired and often did not live after hatching.

The lead compounds studied may be divided into two groups:

Inorganic: Colloidal lead, colloidal lead sulphide, lead phosphate, lead carbonate.

Organic: Lead oleate, lead stearate, lead glyceride, lead lecithin compound.

Of the organic group it is to be noted that they are all fat-soluble. This is intentional, as it has been shown by Bell (*loco citato*) that neoplasms have a high lipin content and by the writer that the lead present in egg yolk is evidently a fat-soluble compound.

Injection Technique.

The technique is essentially that of Tomita,[34] except that iodine and sticking plaster were not used. The injection was performed as follows. The air-lock end of the egg after sterilization with alcohol was drilled with a sterile needle, so as not to puncture the egg membrane. The requisite amount of solution to be tested was then injected from a sterile hyperdermic syringe, the hole in the egg sealed with pure paraffin wax and the egg incubated in the usual manner. Development was followed by "candling" the egg over an electric "spot-light." Before the results obtained are considered the preparation of the various colloidal compounds used will be given.

Preparation of the Lead Compounds.

Colloidal lead was prepared according to Bell's method and as used by the Cancer Research Treatment Committee of this University. In all the solutions prepared gelatine was used in the dispersing medium, the solutions being made isotonic by the addition of Ringer's solution after preparation.

Colloidal lead sulphide: The theoretical amount of 2% sodium sulphide solution was added to a 1% solution of lead chloride in 0·5% solution of gelatine. The resulting solution was centrifuged before injection.

Colloidal lead phosphate was prepared by the addition of trisodium phosphate to lead chloride according to the equation

$$3PbCl_2 + 2Na_3PO_4 = Pb_3(PO_4)_2 + 6NaCl$$

Centrifuging and subsequent analysis showed that the compound was $Pb_3(PO_4)_2$ and that no lead chloride had been carried down by entrainment as suggested by Heintz[21] to form a complex of the form $PbCl_2 \cdot 3Pb_3(PO_4)_2 \cdot H_2O$ and also that no $PbHPO_4$ is present as suggested by Alders and Stähler.[1]

Colloidal lead carbonate: The normal carbonate can be prepared by the addition of sodium bicarbonate to an aqueous lead chloride solution. The theoretical amount of sodium bicarbonate as given by the equation

$$2NaHCO_3 + PbCl_2 = PbCO_3 + 2NaCl + H_2CO_3$$

was added to the gelatine solution of lead chloride. The colloidal solution was centrifuged and used immediately, as it was unstable after one hour.

The question arises as to the possibility of basic lead carbonate also being present, since Sacher[32] states that at room temperature the normal lead carbonate converts slowly to the basic carbonate $PbCO_3 \cdot Pb(OH)_2$. This reaction has been shown by Altmann[2] to be due to the loss of acid from solution and may be kept at a minimum by the use of stoppered flasks. Estimation of the carbonate in the sample prepared by the above method showed that only the normal lead carbonate was present.

Lead oleate and lead stearate were prepared by the addition of the gelatine solution of lead chloride to the sodium salts of the fatty acids, Merck's acids being used. The soaps were prepared by hydrolysis with one-tenth normal sodium hydroxide solution. The resulting colloidal solutions were centrifuged.

Lead glyceride was prepared by bringing a mixture of 30 parts of glycerine, 44 parts lead acetate in 500 cubic centimetres water and 20 parts of potassium hydroxide (all by weight) nearly to boiling, then allowing to stand three to four days, when crystals of the glyceride separate. The compound so prepared had a composition of lead : glycerine = 1 : 1. The solution was prepared by grinding the washed crystals in distilled water-gelatine solution in a glass mortar and centrifuging. A concentration of 0·5 milligrammes of lead per cubic centimetre could thus be obtained.

Lead lecithin compound: A small sample of pure lecithin was prepared by Levene's method.[22] The lecithin was suspended in Ringer-gelatine solution and a solution of lead chloride added, until an equivalent of 9% of lead was present calculated on the lecithin used. No precipitate was formed. For injection this solution was used after centrifuging. Precipitation of the lecithin by addition of acetone and washing with acetone and water mixture gave a product containing 8·45% lead. When commercial lecithin is used, a precipitate is obtained on addition of the lead salt and contains 10·5% to 11·5% of lead and is obviously not a pure lecithin-lead compound owing to the presence of the other phosphatides.

Results of Injection.

The results of the injection experiments show that the toxicity of the lead depends entirely on the compound used. Of those given above the order of toxicity is lead chloride, lead carbonate, colloidal

lead, lead sulphide, lead phosphate, lead stearate, lead oleate, lead glyceride, lead lecithin compound, given in order of decreasing toxicities.

The results of the injection experiments are given in Table IX.

The following points shown by Table IX are of interest.

The embryos which had been injected with lead compounds, had in every case a mean lower wet weight than embryos not injected, that is, than blanks. In this respect lead glyceride, although allowing growth when an equivalent of 0·02 milligramme of lead was injected into the egg, caused a greater depression in the wet weight of the embryo than any of the other compounds tried. It is obvious from the table that lead lecithin compound is the least toxic to the embryo.

With the more toxic compounds more lead is absorbed from a given injection than from the less toxic compounds, although when the amount of these is increased, the amount of lead absorbed is greater than for the toxic compounds, but no ill effects are observed. Thus for lead phosphate it is shown that with an injection of an equivalent of 0·01 milligramme of lead, the amount found in the embryo is 0·161 milligramme per hundred grammes of wet material. When the lead lecithin compound is used, of an injection equivalent of 0·01 milligramme of lead only 0·140 milligramme of lead is found per hundred grammes of wet embryo, but the amount of lead can be increased in the embryo by injection of an equivalent of 0·04 milligramme of lead to 0·214 milligramme per hundred grammes of wet material without apparent toxicity.

The degree of toxicity is thus apparently dependent on the nature of the lead compound used.

The Lead Compounds in Egg Yolk.

Following the indications outlined in an earlier paper[11] and by the foregoing experiments that the fat-soluble lead compounds are the least toxic to the embryo, it was decided to examine the yolk for the presence of fat-soluble lead compounds.

As has already been stated[11] the lead in the yolk is in a form extractable with solvents, those tried with positive results being ethyl alcohol, methyl alcohol, acetone, ether, chloroform, petroleum ether, 5% nitric acid, 5% hydrochloric acid. It is not proposed to give all the extraction experiments in detail; one will suffice.

Ether extraction: Two yolks from eggs of the Dulwich Hill series[11] were dried at 100° C. and extracted with ether in a Soxhelet extracter for forty-eight hours. The ether was removed on a water bath, the fat dried at 100° C.. The fats so obtained were dark red-brown, showing oxidation.

Lead was estimated by the method given earlier in this paper.

The results were as follow: Two yolks, D21 and D22, had a wet weight of 37·2504 grammes, the dry weight (100° C.) was 19·0024 grammes and the water content 51%.

Ether extract was 4·5306 grammes or 12·15% on wet yolks, 23·6% on dry yolks.

The residue in extracter was a white, hard powder. Its dry weight (100° C.) was 14·4686 grammes.

Lead content of fats:

Used for analysis: (1) 2·7108 grammes, (2) 1·8198 grammes.

Lead found: (1) 0·06 milligramme, (2) 0·03 milligramme.

Lead per yolk: (1) 0·05 milligramme, (2) 0·05 milligramme.

Residue used: (1) 14·4702 grammes.

Lead found: (1) Less than 0·005 milligramme.

Hence lead extracted by the ether from the yolk is approximately 99%.

Examination of Lecithin of Egg Yolk.

The wet yolks were extracted with an equal volume of pure acetone by shaking in a large bottle. This process was repeated with fresh quantities of acetone until a colourless acetone extract was obtained. The residual white powder was pressed on a Buchner funnel until it was as free from acetone as possible, then it was transferred to a bottle and shaken with four volumes of chloroform. After decanting the extraction was repeated with four volumes of chloroform and again with two volumes of chloroform. The combined chloroform extracts were concentrated to a syrup under reduced pressure. The almost colourless syrup of crude lecithin solidified on cooling and gave a positive result to a test for lead.

The crude lecithin was dissolved in two to three volumes of ethyl alcohol and a saturated solution of cadmium chloride in ethyl alcohol was added as long as any precipitate was formed.

The precipitate of cadmium chloride salts was purified by Levene's method.[22] After pressing the precipitate was suspended in an equal volume of toluene, when a clear solution resulted on warming. This solution was then poured into four volumes of ice cold ether containing 1% of water. A precipitate formed in a few minutes and was removed by centrifuging. The precipitate was washed with ether twice and then suspended in acetone, centrifuged, again suspended in acetone and again centrifuged to remove adhering toluene.

A sample of lecithin cadmium chloride so produced gave a positive result to a test for lead.

In the analysis 1·4320 grammes of lecithin cadmium chloride were used. The lead found was 0·005 milligramme or 0·00035%.

The lead in this compound is obviously present as a contamination and the problem was to effect a separation of the lead from the lecithin compound.

Many attempts proving fruitless, the steps in the preparation of pure lecithin were carefully repeated.

It was found at the stage prior to the precipitation of the lecithin cadmium chloride compound when the crude lecithin was dissolved in absolute

TABLE IX.

The volume of solution injected into the eggs in the following table was the same for all eggs, that is, 0·25 cubic centimetres. The concentration of lead injected was varied and is recorded as "milligrammes of lead injected" calculated as metallic lead and not in terms of the compounds used.

Lead Compound.	Lead Injected, Milligramme.	Results of Four Eggs.	Embryo.			Milligramme of Lead Found.		
			Age in Days.	Weight in Grammes.		Per Embryo.	Per 100 Grammes of Embryo.	
				Wet.	Dry.		Wet.	Dry.
Lead carbonate ($PbCO_3$)	(1) 0·001	Growth in 3	19	21·6041	4·332	0·025	0·115	0·518
	(2) 0·01	Growth in 2	19	19·5432	4·3514	0·030	0·154	0·690
	(3) 0·02	Nil	—	—	—	—	—	—
	(4) Blank	Growth in 3	19	23·1640	5·2466	0·030	0·130	0·573
Lead colloid	(1) 0·001	Growth in 3	19	22·4826	5·0264	0·025	0·111	0·497
	(2) 0·01	Growth in 3	19	20·0475	4·4650	0·030	0·149	0·672
	(3) 0·02	Nil	—	—	—	—	—	—
	(4) Blank	Growth in 2	19	25·7512	5·6012	0·030	0·117	0·535
Lead sulphide (PbS)	(1) 0·001	Growth in 2	19	20·3462	4·5124	0·030	0·148	0·665
	(2) 0·01	Growth in 3	19	19·3740	4·3046	0·030	0·155	0·696
	(3) 0·02	Nil	—	—	—	—	—	—
	(4) Blank	Growth in 4	19	24·4655	5·4520	0·035	0·148	0·643
Lead phosphate $Pb_3(PO_4)_2$	(1) 0·001	Growth in 3	19	19·3647	4·3320	0·030	0·155	0·695
	(2) 0·01	Growth in 1	19	20·2403	4·5104	0·035	0·173	0·775
	(3) 0·02	Nil	—	—	—	—	—	—
	(4) Blank	Growth in 3	19	23·6254	5·1542	0·035	0·148	0·680
Lead stearate	(1) 0·001	Growth in 4	19	22·4752	5·0041	0·035	0·156	0·700
	(2) 0·01	Growth in 2	19	21·6928	4·8302	0·035	0·161	0·725
	(3) 0·02	Nil	—	—	—	—	—	—
	(4) Blank	Growth in 3	19	24·4263	5·4577	0·035	0·143	0·643
Lead oleate	(1) 0·001	Growth in 3	19	23·6124	5·2754	0·030	0·127	0·570
	(2) 0·01	Growth in 3	19	22·4275	5·0026	0·033	0·147	0·660
	(3) 0·02	Growth in 1	19	20·6290	4·6013	0·045	0·218	0·980
	(4) 0·04	Nil	—	—	—	—	—	—
	(5) Blank	Growth in 3	19	24·1749	5·3842	0·035	0·145	0·650
Lead glyceride	(1) 0·001	Growth in 2	19	19·7546	4·4424	0·028	0·142	0·630
	(2) 0·01	Growth in 1	19	19·2430	4·3072	0·030	0·156	0·697
	(3) 0·02	Growth in 1	19	17·2043	3·8421	0·030	0·174	0·782
	(4) 0·04	Nil	—	—	—	—	—	—
	(5) Blank	Growth in 4	19	24·7540	5·5124	0·034	0·137	0·617
Lead lecithin compound	(1) 0·001	Growth in 4	19	24·4290	5·4513	0·034	0·139	0·624
	(2) 0·01	Growth in 3	19	23·6752	5·2742	0·033	0·140	0·626
	(3) 0·02	Growth in 3	19	24·2043	5·4037	0·040	0·165	0·740
	(4) 0·04	Growth in 1	19	23·3202	5·2044	0·050	0·214	0·960
	(5) 0·06	Nil	—	—	—	—	—	—
	(6) Blank	Growth in 3	19	25·2763	5·6427	0·033	0·130	0·585

Injections were made with lead chloride solution, but no growth was observed even with 0·001 milligramme of lead.

alcohol, that the solution so obtained was never clear, a slight opalescence always existing. This had been disregarded. However, on centrifuging a clear solution was obtained together with a light brown amorphous precipitate. From the clear alcoholic solution of the crude lecithin a lead-free cadmium chloride lecithin compound was obtained.

The brown precipitate was found to contain lead. Attempts to crystallize the compound have been fruitless, but reprecipitation from chloroform solution has produced a compound of constant composition.

The procedure therefore adopted for the separation of the lead compound from the lecithin solids of the egg yolk was as follows: (See also Figure XII.) The crude lecithin obtained as previously mentioned was dissolved in two to three volumes of absolute ethyl alcohol, well mixed and allowed to stand five minutes in the cold. The slightly opalescent solution was then centrifuged until a clear supernatent fluid resulted, usually after ten minutes at a speed of three thousand revolutions per minute. The clear supernatent liquid was then decanted and the precipitate washed with two volumes of absolute ethyl alcohol (cold), again centrifuged and decanted. The light brown powder was then dissolved in chloroform, the least amount possible being used, and absolute alcohol added until the powder was reprecipitated. This process was repeated. The resulting powder, air-dried, was a dark cream colour, very granular, but apparently non-crystalline.

Following the scheme shown in Figure XIV, 1,750 yolks freed from white were treated and from the 31·5 kilograms of wet material 0·0106 gramme of twice precipitated lead compound containing 11·6% lead was obtained.

The analysis of the lead compound obtained from the lecithin solids of egg yolk was as follows:

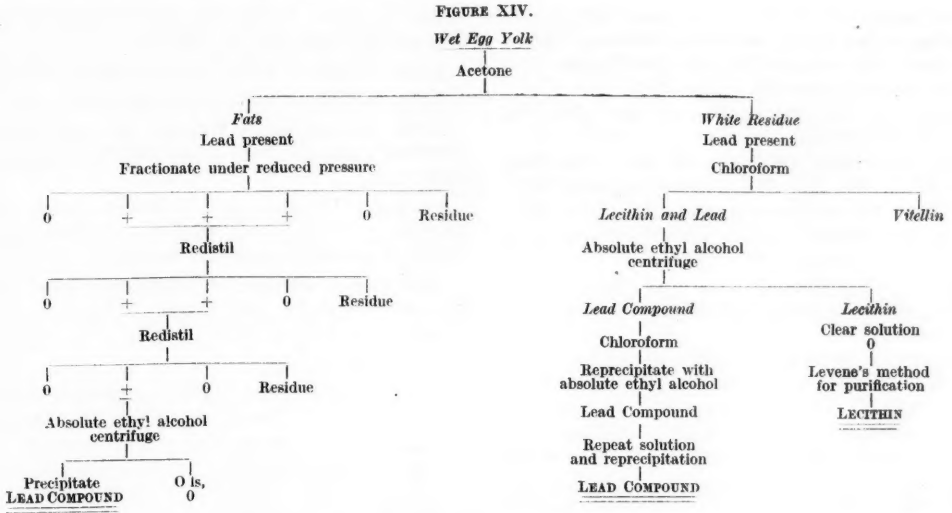
Light brown powder, twice precipitated from chloroform by addition of absolute alcohol.

Used 1,750 yolks, wet weight 31·5 kilograms.

Brown powder recovered after two reprecipitations, 10·63 milligrammes.

Note that in the following analyses the micro-methods of Fritz Pregl as given in his book, *"Die*

FIGURE XIV.



0 = no lead. + = lead present.

Quantitative Organische Mikroanalyse," Second Edition, were followed.

Lead: Weight of lead compound used, 0·001830 gramme; lead found, 0·212 milligramme or 11·584%.

Carbon: Weight of lead compound used, 0·003475 gramme; weight of carbon dioxide produced, 0·007364 gramme; carbon, 57·78%.

Hydrogen: Weight of water produced, 0·002995 gramme; hydrogen, 9·57%.

Nitrogen (Micro-Dumas method): Weight of substance used, 0·002291 gramme; barometer, 759·0 millimetres; temperature, 20° C.; volume of nitrogen, 0·0315 cubic centimetre; nitrogen, 1·60%.

Phosphorus (Bell-Doisy): Weight of compound used, 1·4760 milligrammes; phosphorus found, 0·0502 milligramme; phosphorus, 3·40%.

Summary:

	Found	Calculated for $[C_{43}H_{86}O_9NP]_2Pb$
	%	%
Carbon	57·78	57·68
Hydrogen	9·57	9·61
Nitrogen	1·60	1·56
Phosphorus ..	3·40	3·47
Lead	11·58	11·57
Oxygen	16·07 (by difference)	16·10

The remainder of the compound (1·5 milligrammes) was hydrolysed by boiling with 3% sulphuric acid for eight hours as detailed by MacLean.[23a] Subsequently Levene's[22a] procedure was followed. As such a small amount of substance was available recrystallizations were not attempted. Under the microscope definite needle crystals of the picrate were observed, which melted at 243° C., comparing favourably with that given by Levene for choline picrate of 241° to 242° C. (uncorrected). Conversion of the picrate to the platinum salt was not successfully achieved, as too little for analysis resulted. The fatty acids resulting from the hydrolysis could not be investigated in detail, other than that the mixture was unsaturated and melted at 58·5° C..

However, with the foregoing data it seems most probable that the lead compound isolated is a lead lecithin compound, consisting of two molecules of lecithin and one molecule of lead, namely $[C_{43}H_{86}O_9NP]_2Pb$.

Examination of Fats.

The acetone extract of the egg yolks from which the lecithin and lead compound were prepared, was concentrated under reduced pressure and dried *in vacuo* at 40° C. over calcium chloride to prevent oxidation of the fats. The fats so obtained were light golden yellow in colour, while when the fats were dried at 100° C., they were red-brown in colour.

Following the scheme of Figure XIV the fats were fractionally distilled at 2·5 millimetres pressure, a "Hyvac" mechanical pump being used. Owing to the high temperatures reached in the distillation and also to prevent any contamination from rubber stoppers, an all glass flask with connexions was made from a Claisen flask. A tube of thin walled glass closed at one end to carry the Anschütz thermometers was invaginated into the neck of the flask, contact being made between the thermometer and the glass walls of the tube by means of a few drops of mercury. This arrangement also permits of rapid change of thermometers without interrupting the distillation. The capillary boiling tube was made to fit roughly the other arm of the flask and sealed by an external sleeve of thin rubber tubing.

The distillation of the fats was very difficult at the beginning owing to the excessive frothing. After about an hour this ceased and the distillation proceeded in a normal manner.

The results of the fractionations are given in Tables X, XI and XII.

The lead compound in the yolk fats was thus concentrated in the fraction boiling between 205° and 210° C. at 2·5 millimetres pressure. This fraction was a pale yellow, solid, non-crystalline with no characteristic odour. The volume of this fraction from 6·4 kilograms of fats was thirty cubic centimetres. Melting point and refractive index are given in Table XI.

On treating a portion of this fraction with absolute ethyl alcohol an emulsion formed, persisting when warmed. After standing half an hour the appearance changed and a slight settling was observed. Centrifuging produced a clear solution and a white granular solid, insoluble in alcohol, but soluble in ether and chloroform. Results of tests for lead were positive for the solid, only traces being shown in the remaining fats.

The remainder of the fraction was then treated with six volumes of hot absolute ethyl alcohol and allowed to stand half an hour. It was then centrifuged. The white granular precipitate was washed three times with hot absolute alcohol and then air dried. From chloroform the solid did not crystallize, but separated as a white, transparent, waxy material.

The analysis of the compound isolated from the fats of egg yolk was as follows:

Total weight of solid obtained from 6·4 kilograms of fats, 0·2860 gramme; melting point, 79·5° C..

Lead Estimation: Weight of substance used, 0·001095 gramme; lead found, 0·294 milligramme or 26·86%.

Carbon: Weight of substance used, 0·002885 gramme; weight of carbon dioxide produced, 0·005948 gramme; carbon, 56·23%.

Hydrogen: Weight of substance used, 0·002885 gramme; weight of water produced, 0·002222 gramme; hydrogen, 8·56%.

Summary:

	Found	Calculated for $(C_{17}H_{33}COO)_2Pb$
Carbon ...	56·23%	56·38%
Hydrogen .	8·56%	8·58%
Oxygen ..	8·41%	8·32%
Lead	26·86%	26·92%

Examination of Fatty Acid.

The remainder of the substance was dissolved in benzene and saturated with sulphuretted hydrogen and allowed to stand over night. The lead sulphide was removed by centrifuging and the excess sulphuretted hydrogen and solvent by evaporation in a vacuum desiccator at 40° C.. The remaining light yellow oil was dried at 95° C. in an air oven.

TABLE X.

Number.	Boiling-point Range.	Melting-point of Distillate.	Refractive Index (Abbé).	Result of Lead Test.
1	To 170° C.	Aqueous oily mixture	—	—
2	170°–180° C.	37·6° C.	1·4531 (38° C.)	Not present
3	180°–200° C.	34·4° C.	1·4557 (38° C.)	Present
4	200°–220° C.	30·2° C.	1·4568 (38° C.)	Present
5	220°–245° C.	34·6° C.	1·4683	Present
6	245°–260° C.	41·2° C.	1·4644	Not present
7	260°–300° C.	42·6° C.	1·4613	Not present
	Residue	Dark brown with some charring	—	Not present

Original oil: Light yellow, liquid; R.I. (Abbé), 1·4623 at 38° C.

TABLE XI.

Refractionation of Fractions 3, 4 and 5 containing Lead.

Number.	Boiling-point Range.	Melting-point of Distillate.	Refractive Index (Abbé).	Result of Lead Test.
1	To 200° C.	Few drops	—	Present
2	200°–210° C.	30·8° C.	1·4556 (38° C.)	Present
3	210°–220° C.	31·6° C.	1·4565	Present
4	220°–230° C.	32·8° C.	1·4567	Traces present
5	230°–240° C.	33·1° C.	1·4564	Not present

TABLE XII.

Refractionation of Fractions 2 and 3 containing Lead.

Number.	Boiling-point Range.	Melting-point of Distillate.	Refractive Index (Abbé).	Result of Lead Test.
1	200°–205° C.	30·6° C.	1·4556 (38° C.)	Not present
2	205°–210° C.	31·2° C.	1·4564	Present
3	210°–215° C.	31·4° C.	1·4566	Trace
4	215°–220° C.	31·8° C.	1·4566	Not present
	Residue	Viscous dark brown oil	—	Not present

The analysis was as follows:

Carbon: Acid used for analysis, 0·002467 gramme; carbon dioxide produced, 0·006900 gramme; carbon, 76·46%.

Hydrogen: Water produced, 0·002710 gramme; hydrogen, 12·21%.

Summary:

	Found	Calculated for $C_{18}H_{34}O_2$
Carbon	76·46%	.. 76·59%
Hydrogen	12·21%	.. 12·05%
Oxygen (by difference)	11·33%	.. 11·35%

The acid was unsaturated as shown by bromine absorption.

Refractive index (Abbé), 1·4613 at 20° C..

Refractive index of oleic acid, 1·4620 at 20° C. (Procter).

Melting point of the acid, 14·4° C. (uncorrected).

Melting point of pure oleic acid, 14·30° C..

When the foregoing analyses are considered, it seems certain that the lead compound extracted by fractioned distillation from egg yolk is lead oleate.

That the lead represented by the two compounds extracted and reported in this paper is practically all the lead in the yolks treated is seen from the following considerations. Previous lead determinations for yolks of eggs in the same series as were employed in this paper showed a mean lead content per yolk of 0·05 milligramme.

Now 10·63 milligrammes of the lead lecithin compound were recovered, equivalent to 1·23 milligrammes of lead, as analysis has shown that the compound contains 11·6% lead.

Of the lead oleate 0·2860 gramme was recovered which, containing 26·86% lead, represents 0·0765 gramme of lead.

Thus the total lead represented by the amounts of the two compounds extracted from 1,750 yolks is 0·07773 gramme or 0·044 milligramme per yolk.

It is thus seen that of the possible lead obtainable from the yolks, 88·5% is accounted for, which can be considered quite satisfactory when it is remembered that the final material suffered losses due to purifications for analysis.

SUMMARY.

1. In this paper have been presented data concerning eggs from the same fowl, namely, the weights of eggs, the loss of weight during incubation and the weights of embryos.

2. The absorption of lead from various compounds by the developing embryo has been briefly noted, also the relative toxicity of the compounds used.

3. The absorption of the lead naturally present in the egg by the developing embryo has been studied from six to twenty days for eggs from two fowls.

4. The nature of the lead compound in the egg yolk has been studied. The isolation of two compounds is reported, namely, lead oleate, occurring in the extractable portion of the yolk to the extent of 0·001% on the wet yolk, and a lead lecithin compound, occurring in the lecithin solids of the yolk to the extent of 0·00004% on the wet yolk.

ACKNOWLEDGEMENTS.

The author has much pleasure in acknowledging his indebtedness to Professor H. Priestley for his help and criticism of this paper; to Professor Earl, of the Organic Chemistry Department, who placed the apparatus used for the microanalyses at his disposal and for his assistance in using the microbalance; and to Dr. H. G. Chapman, Director of Cancer Research, for his helpful criticism and advice during the course of this investigation.

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MOLLUSCUM CONTAGIOSUM AND TURKISH BATHS.

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THE infectivity of *molluscum contagiosum* is a well established fact. Contagion by the inoculation experiments has demonstrated this on many occasions, although the causative virus, if such it be, has never been isolated. This disease is one of those very rarely met with in Australia, whatever the reason may be and therefore the incidence of three cases within two days, recently seen in Melbourne, is very unusual. All conformed to the typical picture and presented the small round tumour lesions of a pinkish, translucent, waxy nature, varying in size from a pin's head to that of a small pea; the larger ones had the typical depressed centre seen in cases of *molluscum contagiosum*. Distribution in each case was on the chest and abdomen. The first patient, a male, was rather concerned about three or four small furuncles. The lesions had become pus infected. The second patient, seen on the same day, was a patient of Dr. Colquhoun at the Repatriation Hospital and in this instance treatment was sought for a condition not associated in any way with *molluscum contagiosum*. The lesions had been present for some time and little notice taken of them. This may partly explain the reason why so few examples of this disease come under observation. There are no subjective symptoms if there is not superimposed infection and it is possible in some instances that the presence of small tumours obviously quite superficial is not in the patient's opinion a sufficient reason for seeking medical advice.

The third patient in this series, seen on the following day, also presented the classical signs. Each of the three was asked the usual question, whether he had been taking Turkish baths a week or so before the eruption appeared, and all answered this had been their practice. Moreover, it was further elicited that the same baths had been attended by each patient.

The proprietor of the baths was duly informed of the facts and warned of the necessity of boiling towels, mats and similar material and of treating the slabs with steam and disinfectant. This small series, briefly recorded, is interesting from the fact of the source of contagion having been traced to Turkish baths at the same bathing establishment and so positively proves the contention of Malcolm Morris, Crocker and Hutchinson, who drew attention to the association of *molluscum contagiosum* and Turkish baths in England.

FRACTURES OF THE LOWER END OF THE HUMERUS IN CHILDREN.

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FROM patients seen at the Children's Hospital and from discussions with practitioners it is evident that the teaching of Sir Robert Jones on the treatment of fractures about the elbow joint is yet not fully understood. The method as used at Liverpool for all fractures around the elbow, except fracture of the olecranon, is as follows. After manipulative

reduction by hyperextension, supination and traction, followed by acute flexion, the limb is suspended from the neck by what is known as the "cuff and collar" method. This position is several degrees short of full flexion. No bandage nor strapping of any kind is applied to the limb other than the cuff around the wrist. The limb is kept in this position for three weeks, during which period it is left severely alone. At the end of this period the sling is lowered 2·5 to 5·0 centimetres (one to two inches) and voluntary movements encouraged within the range thus allowed, but without release of the sling. If in three days the forearm can be raised voluntarily to its previous position of acute flexion, the sling is lowered further. This process is repeated every few days, always provided that the original position is voluntarily obtained. When the forearm has been lowered to past the right angle, the sling may be dispensed with and the arm given full freedom. The remaining extension will gradually return. No attempts should be made to hurry this by passive movements under any consideration. During the period that the sling is being gradually lowered, any failure to regain the position of acute flexion is an indication for a further period of rest in this position. Failure to act on this indication is responsible for a great number of bad results and the fear that full extension will not be regained, with the ensuing meddlesome surgery in attempts to hurry this extension, accounts for many more. It cannot be too definitely taught that if flexion is insured by the above methods, extension may take care of itself. The "cuff and collar" consist of two lengths of tubing of soft leather. A bandage is passed through the cuff and this is tied around the wrist. The long end of the bandage is then passed through the collar which is placed around the neck and tied so that the wrist is well up under the chin, the ball of the thumb being against the side of the neck. I have had opportunity recently, over a period of fifteen months, of observing great numbers of these fractures treated by this method at Liverpool and the results are remarkably good. Moreover, in the orthopædic clinics there (at which all fractures are treated) very few stiff elbows are seen either in adults or children, considering the great numbers dealt with. This is only accounted for by the fact that the above methods have been taught and universally followed for nearly forty years. The concluding paragraph of Sir Robert Jones's original communication on this matter, written in 1894, still holds good.

If these principles are adhered to anchylosis will prove a rare result. Sometimes the joint falls short of a few degrees of complete extension; but I am fully persuaded that the best results in the greatest number of cases can only be realized by extending and supinating the arm and acutely flexing the elbow.

It is because of the frequent bad results still obtained after these fractures, results which could be avoided if this simplest of treatment were faithfully carried out, that this communication is written.

Reports of Cases.

TWO CASES OF SUDDEN DEATH: FROM RUPTURE OF THE AORTA AND FROM RUPTURE OF AN ABDOMINAL ANEURYSM.

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AND

JOHN FIDDES, M.D. (Aberdeen).

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UNEXPECTED sudden death of an individual is of great importance generally, medico-legally and clinically. When such an occurrence happens in persons who have never complained of ill health, it comes as a shock to friends and relatives and the physician can rarely certify with assurance what factors contributed to the tragedy. Two such cases which occurred within a fortnight, we considered to be of sufficient interest and rarity to warrant publication.

Case I.

A labourer, aged fifty-eight years, while giving an exhibition of his muscular strength in relation to the task at which he was employed, was seized suddenly with præcordial pain and extreme weakness, causing him to fall. According to the testimony of those present he was unconscious for several minutes. The weakness persisted and was accompanied by nausea. The patient said he had never been ill before and that his general health had been excellent.

On examination he was found to be in a state of extreme collapse with cyanosed face and cold, clammy skin. His tongue was furred and dry and his lips were dry. The pulse was regular and the heart sounds, although faint, were apparently normal. The patient died four hours after admission to hospital.

Autopsy.

The subject was a well developed, middle-aged man, 1·8 metres (six feet) in height. The pericardium contained about one and a half litres of blood and there was an extravasation of blood into the subpericardial tissue over the aorta. The aorta was opened up and it was seen to be torn transversely completely through *intima* and *media* (see Figure I). The tear was four centimetres in length and was situated three centimetres above the aortic valve. The aorta in the neighbourhood of the tear appeared to be quite normal. Just above the sinuses of Valsalva and round the orifices of the innominate left carotid and left subclavian arteries there were many raised yellow and grey plaques. There was no wrinkling or scarring of the *intima*. The circumference of the aortic ring was 8·2 centimetres. At the tear the aorta was 9·9 centimetres in circumference, while at the orifice of the innominate artery it was 8·5 centimetres in circumference. The distance of the innominate arteries from the aortic valve was 9·7 centimetres.

The heart weighed 410 grammes. The following measurements were made: Circumference of mitral valve, 9 centimetres; circumference of tricuspid valve, 9 centimetres; circumference of aortic valve, 8·2 centimetres; circumference of pulmonary valve, 7 centimetres. The myocardium of the left ventricle varied from 1·2 centimetres to 1·8 centimetres in thickness, while that of the right ventricle varied from 0·4 centimetre to 0·7 centimetre.

There were no apparent abnormalities of the endocardium. The myocardium was slightly softer in consistency than normal. The coronary arteries appeared to be normal. There was nothing of pathological interest found in the other organs.

Microscopically the aorta showed the following changes. In sections through the atheromatous areas there was the usual laminated layer of fibrous tissue cells covering a deeper structureless area adjacent to the *media* and staining deeply with Sudan III. The smooth muscle and con-

nective tissue layers of the *media* were uniform and uninterrupted. The elastic tissue, stained by Veroff's method, was evenly distributed and normal in amount. The *vasa vasorum* adjacent to the tear showed slight perivascular fibrosis and there was a small accumulation of polymorphonuclear leucocytes and mononuclear cells. The adventitia contained extravasated blood. Frozen section of the myocardium stained with Sudan III showed fatty infiltration with slightly fatty degeneration of the muscle fibres.

FIGURE I.

Case I. Heart and aorta showing tear and blood extravasation into surrounding tissues. The tear marked TT was cut through by the incision exposing the aorta.

Case II.

A man, aged sixty-two, was quite well until six weeks before admission to hospital. Since then he had complained of an aching pain across the sacrum and hips and lower part of the abdomen. On one occasion there was a shooting pain down the right leg. The patient had been a heavy drinker. There was no history of previous illness.

On examination the patient was a sallow, middle-aged man, looking moderately ill and having frequent attacks of pain, causing him to draw up his knees and grasp his abdomen. His pupils reacted to light and accommodation. The pulse was regular, its rate was 120 per minute. The arteries of the extremities were not noticeably thickened. The systolic blood pressure was 158 millimetres of mercury. The apex beat of the heart was in the fifth left interspace, 7·5 centimetres from the mid-line. There was no right cardiac dulness. The heart sounds were regular and there were no bruits. The lungs were hyperresonant to percussion, otherwise no abnormality was detected.

The abdomen moved well on respiration and there was no rigidity. There were two finger-breadths of liver dulness below the costal margin. No free fluid was detected. There was slight tenderness in the left hypochondrium. No mass could be palpated.

Examination of the nervous system showed that the knee jerks were equal and active; the plantar reflex was flexor; the superficial abdominal reflexes were present.

The specific gravity of the urine was 1022; it was acid and contained neither albumin nor sugar. General microscopical examination revealed no abnormality.

The Wassermann test was negative. Barium examination for carcinoma of the bowel revealed no abnormality.

There was no change in the symptoms and on the eighth day after admission the patient died suddenly while lying in bed.

Autopsy.

On opening the abdomen there was a large quantity of blood clot in the peritoneal cavity. There was a subperitoneal swelling at the site of bifurcation of the abdominal aorta and at the lower part of the swelling the peritoneum was torn over an ulcerated area about one centimetre in diameter, with an irregular floor composed of blood clot. On section the swelling was seen to be an aneurysm of the abdominal aorta at its termination, involving also both common iliac arteries and it had perforated into the peritoneal cavity (see Figure II). The aneurysm was of the saccular type and the wall was composed of laminated clot with surrounding fibrosis. The abdominal aorta manifested yellow and grey plaques and there were similar thickenings of the *intima* of the thoracic aorta. The aortic valve (circumference seven centimetres) was competent to the water test. The coronary arteries were atheromatous. The heart weighed 310 grammes. The myocardium was firm in consistency and the mitral and tricuspid valves were apparently normal. The left lung was adherent to the chest wall and there were scarred areas at the apex. The right lung was emphysematous and also showed apical scarring. The liver weighed 1,270 grammes. It was pale in colour and soft in consistency. The right and left kidneys weighed 140 grammes and 150 grammes respectively. In both the cortex was of normal width and well demarcated from the medulla. The arterioles on the cut surface were gaping and had thickened walls. The capsule was slightly adherent. Examination of the renal arteries revealed hypertrophy of the *media*. The other abdominal organs and the alimentary tract presented no abnormalities.

Microscopically the *intima* of the aorta was thickened in patches with areas of atheromatous degeneration. The *media* and *adventitia* showed no pathological change. The liver was in a condition of fatty degeneration, this being most pronounced at the periphery of the lobules. Degeneration was not prominent in the kidney, although there were some thickening of the arterioles and slight degeneration of the tubular epithelium and glomeruli. The capsule showed some thickening in parts. The heart muscle appeared normal, no lesion being found after extensive investigation.

Discussion.

The two cases of sudden death here recorded occurred under very dissimilar conditions. In one the man, a labourer, was exhibiting muscular prowess, using the utter limit of his physical powers, while the other patient died suddenly in the position of repose, in bed. In healthy young people the normal vessel wall can accommodate itself readily to altered pressures practically with impunity, but in the artery weakened by chronic inflammation and degeneration a dilatation or a rupture under the strain of a sudden increase in blood pressure is quite an expected occurrence. The unusual feature in these two cases is the absence in any pronounced degree of such pathological change. In Case I the microscopical examination revealed no pronounced degeneration of the *media* and the measurements exclude any undue dilatation of the aorta; in Case II also the *media* was practically unaffected except at the site of the aneurysm.

Men used to continuous hard manual labour often suffer from hardening of the arteries. Clifford Allbutt[(2)] points out that arteriosclerosis is often confined to the vessels of

the limbs used and attributes it to the fact that the arteries suffer from a large, swift output, telling upon them when in a state of relaxation and that this in the long arteries of the limbs may produce Monckeberg's form of arteriosclerosis which, while deforming the vessel, need not necessarily signify disease of the vessels of other parts, where, indeed, the pressure may be proportionately relieved.

In the first case here reported there was no lesion to be found in the part of the artery where the rupture occurred, except the slight fibrosis around the *vasa vasorum*. Clifford Allbutt[2] writes:

> Rupture of an aortic aneurism is common, but rupture of an aorta apparently sound, or in a comparatively young person, if an infrequent, is not now a surprising event.

He mentions several well authenticated cases.

Case II was in sharp contrast to Case I. Here the systolic blood pressure was 158 millimetres of mercury, which is not excessively high for a man of sixty-two years. Clifford Allbutt separates arterial lesions into the high pressure type and the decrescent type and states that arteriosclerotics with moderate pressure often attain great length of years and as a class live longer than those of the high pressure type. Case II appears to have been more like the second group, but the possibility of a long life was cut short by an unusual complication. Here again the *intima* of the large arteries manifested some atheromatous degeneration, but there was no evidence of mesaortitis and extensive microscopical investigation revealed no gummatous changes anywhere. The Wassermann test was negative. Clinically the condition was a difficult one to diagnose, the patient being a man with a good history regarding health up to the time of the onset of his last and fatal illness.

FIGURE II.
Case II. Abdominal aorta showing aneurysm at site of bifurcation. The situation of the perforation is marked X.

The pain experienced suggested inflammatory or pressure symptoms. X ray examination for bowel tumour was reported as disclosing no abnormality. Examination of the urine revealed nothing pointing to disease of the urinary tract. As the arteries of the limbs showed only slight hardening and the Wassermann test yielded no reaction, the presence of aneurysm did not force itself as a likely cause of the patient's symptoms. The symptoms produced by the presence of the aneurysm were not noticed until six weeks previous to his admission to hospital. From this we may consider the condition to have been a progressive one. Had the sudden collapse occurred during some muscular effort it would have suggested some circulatory disturbance, but its sudden onset while the patient was at rest in bed tended to be misleading. It is well known that the blood pressure in the limbs decreases on assuming the reclining posture and mounts quickly on regaining the erect position, possibly through the sudden constriction of the great splanchnic arterioles.

Various observers have found the blood pressure lowered in sleep often by fifteen to thirty millimetres of mercury. Brook and Carrol[3] in hypertonic subjects found it lowered by forty-four millimetres of mercury. MacWilliam[4] found that there were two entirely different conditions in sleep: (i) sound sleep with lowered pressure and (ii) disturbed sleep, as with dreams *et cetera* which may be attended by remarkable elevations in pressure, for example, systolic pressure raised from 125 millimetres to 182 millimetres of mercury. This was more than he found in the same subjects when exercised as by riding a bicycle, walking *et cetera*.

The man (Case II) was stated to have been a heavy drinker. The effect of continued excess in alcohol consumption is known to have a deleterious effect on the vessels. Clifford Allbutt states:

> In beer drinkers and gluttons the arteries, whether by the bulk of intake or by toxic influences on the kidneys, suffer sooner or later more severely and this deterioration, even on small increments of age, tends like other defects to multiply itself, for as the arteries stiffen in ordinary persons after the age of 60, a muscular effort may send the systolic pressure twice as high as in men of 20-30 years whose vessels are normal.

It is evident that the patient in Case II had stiffened arteriosclerotic vessels and the probability is that some psychic factor caused a sudden increase in blood pressure with consequent rupture of the aneurysm and fatal hæmorrhage.

Acknowledgement.

We take the opportunity here of expressing our thanks to Dr. C. S. Barbour and Dr. Glen Knight for their kindness in supplying the clinical notes.

References.

(1) Clifford Allbutt: "Diseases of the Arteries Including Angina Pectoris," Volume I, page 209.
(2) Clifford Allbutt: "Diseases of the Arteries Including Angina Pectoris," Volume II, page 206.
(3) Brooks and Carrol: *Archives of Internal Medicine*, August 15, 1914.
(4) MacWilliam: *Quarterly Journal of Experimental Physiology*, Volume XIII, 1923.

Reviews.

CLINICAL DIAGNOSIS.

To fill these three large volumes with original articles by many authorities on bedside diagnosis would at first sight appear to be difficult. Dr. Blumer, however, has attempted it and to a great extent succeeded.[1] To say that his books deal solely with bedside diagnosis is surely

[1] "Bedside Diagnosis," by American Authors; Edited by George Blumer, M.D.; in three volumes; 1928. Philadelphia: W. B. Saunders Company. Melbourne: James Little. Royal 8vo., pp. 2820, with illustrations. Price: £6 15s. net.

a stretch, as the many writers who have joined with him, have seldom confined themselves merely to diagnosis. Thus Dr. Richard P. Strong in an absorbing article on plague has discussed every aspect of the question save treatment. Naturally such a method does not make for compactness and the final result is given in three volumes of nearly 900 pages each, covering almost the whole of medicine except treatment and more than one author gets in a few words on that subject.

Dr. Blumer has summoned a most imposing concourse of collaborators. Dr. Strong has already been mentioned; among the sixty odd we find such names as Amos, Blackfan, Emerson, Landis, Musser, Park, Pemberton, Talbot, Edward Jenner Wood (the sole English contributor) and Zingher.

The articles themselves are on the whole excellent; some like that of Dr. Strong lose their very excellence by their diffuseness, while others, like that of Dr. Francis G. Blake on pneumonia, are models of succinct description.

It must be understood that the author of the work presupposes a diagnosis made before the book is consulted. It is a work entirely different from, say, Saville's "Clinical Medicine" or French's "Index."

Thus the doctor who has made a diagnosis of congenital syphilis, may turn to an excellent article obviously written by two men—Fred Wise and Howard J. Parkhurst—who are in daily contact with their subject. Still it is not until he has read thirteen pages of excellent discussion that he comes to the differential diagnosis of the skin conditions which is just what the ordinary man most wants, and then more descriptive writing till he meets the one and a half pages (out of twenty-four) devoted to diagnosis.

Again take congenital heart disease. Here there is a monograph by Maude Abbott and Edgar Wiess which goes more fully into the mechanism of the disease than any article in any other text book we know. The writers hold that until the physician understands the mechanism of the circulation through the fœtal, normal or malformed heart, he cannot hope to make a satisfactory diagnosis. As a separate monograph this section would make a small and admirable book that no physician, especially one interested in diseases of children, could be without. But in these volumes it is buried.

The sections on tropical medicine are models, although as previously stated, those of Dr. Strong err on the side of prolixity. Curiously enough the article on hookworm is meagre and disappointing. The study of sprue by Edward Jenner Wood is very complete, especially the close parallel which he draws between sprue and pernicious anæmia; at the time of writing, however, he did not seem to have knowledge of the even closer relation suggested by the excellent results obtained from liver feeding in sprue.

The chapter on pulmonary tuberculosis by Lawrence Brom and Fred. H. Heise is sound throughout. The writers insist on the use of simple physical means—eye, hand, ear (especially) and nose. At the same time, however, they insist that the only certain diagnostic measure is bacteriological and urge the constant mutual control of physical signs and radiographic records. Otherwise the article contains little that is new, but all that is given is sound.

There is unfortunately no discussion of that most difficult condition "soldiers' lung," post gas or post bronchitic, which may shade off quite imperceptibly into tuberculosis on one side and emphysema and asthma on the other. In such cases the correct diagnosis calls for the greatest possible skill and even the most experienced clinicians may be brought to shame by the revelations of time as the disease progresses.

The sections dealing with difficult aspects of cardiovascular disease are preceded by a detailed study of "Instruments: Aids of Precision in Cardiovascular Diagnosis" by William J. Kerr. The radiograph, the polygraph and the electrocardiograph are described in moderate detail (though it was disappointing to find no comment on the new portable pattern of the electrocardiograph) and the nature of their records explained clearly and shortly. But the author insists that except in special circumstances the use of these instruments is unnecessary and that almost every diagnosis can be made by the eye, ear and finger of experience.

The sphygmomanometer is in a different class as the conditions which it reveals can only be determined by its employment with accuracy; and these include (for example, *pulsus alternans*) conditions of the greatest moment to the patient.

The section on *angina pectoris* seemed disappointing when compared to Mackenzie's work and the diagnosis between true paroxysmal *angina pectoris* and coronary blocking deserves much more than is given. The outlook in the two conditions and the treatment are essentially different and are completely dependent on an accurate diagnosis.

But perhaps one of the best sections in the book is that on diseases of the stomach and small intestine. It is written by Dr. A. L. Holland and his attitude is best illustrated by a quotation:

> It is in a more fundamental way that the X ray has brought about changes in medicine—not all of which are desirable. The neglect of the older diagnostic methods in which history and physical examination were the framework may be somewhat compensated for in speed and accuracy. But the dwindling need for deductive reasoning in this kind of diagnosis can only result in intellectual deterioration.
>
> It would seem, therefore, not entirely out of place at this time to discuss the problems of diagnosis without reference to roentgenology—ignoring it for the time as one of the necessary diagnostic procedures—but utilizing the information it has given concerning the vagaries and normal behaviour of the digestive apparatus.

For eighty-five lucid pages he follows this method and this section more closely conforms to the title of the book than do most of its other chapters.

As is usual in American text books, a large amount of space is devoted to the endocrine diseases and, as is also usual, illustrations are used freely. Among the various types of endocrine disturbance described that on thyreoid disturbance is the best. Most of it is based on the work of the Mayo Clinic and it is clear, graphic and complete. One important point in the diagnosis of endocrine disturbances insisted on by the author is radiography to determine the "bone age" of the patient.

Probably the most disappointing portion of the work is that dealing with the nervous system. It is generally very diffuse; there is an introduction on the anatomy and physiology filling 250 pages and there are some curious omissions. The general and local diseases of the central nervous system are discussed thereafter in sections that vary greatly in value. Thus post encephalitic states, often wrongly diagnosed, are discussed in a few short paragraphs, in spite of the increasing frequency of the condition, its relation to epilepsy, feeble mindedness, moral changes and paralyses. The relationship of combined spinal sclerosis to pernicious anæmia is mentioned with no great emphasis and there is no suggestion of the outlook with liver feeding nor of the Price Jones curve in patients not obviously anæmic. The section on neurosyphilis is full, clear and well arranged. The sections on hysteria and neurasthenia were singularly unhelpful when appealed to in a case of difficult diagnosis between malingering and hysteria in a girl of fourteen.

A few diseases were looked for and not found, for example, pink disease neither as pink disease, erythrœdema nor acrodynia. It is mentioned only in the diagnosis from pellagra. Rat bite fever is not discoverable, but trench fever is. Lead poisoning among children is not mentioned, though the disease among "crib biters" has been described in the United States.

On the whole the work is one which would be of great value to a busy practitioner; the omissions and curtailments are not serious and the best articles deal with diseases of most common occurrence.

The paper is heavy and glossy, the type is clear (though there are a few misprints) and the covers have that green hue so seductive to the domestic cockroach. The separate index volume is complete, accurate and very convenient.

The Medical Journal of Australia

SATURDAY, JUNE 15, 1929.

The Treatment of Snake Bite.

ALTHOUGH snakes are common in Australia and several venomous species are included in the Australian list, the number of deaths from snake bite is relatively small. The average number of deaths each year from this cause is about thirteen in the whole Commonwealth. This represents slightly more than two persons among every million. Moreover, many of these deaths have been due to foolhardiness on the part of showmen who at times claim to be immune to the effects of venom or to possess some secret remedy. Some of the deaths have followed snake bites that could have been avoided by the exercise of reasonable care. Notwithstanding these considerations, the medical profession has a distinct duty to the public in regard to this danger to life. Every medical practitioner should be able to treat snake bite when it occurs, not by applying popular remedies or makeshift procedures, but in accordance with scientific data. Before the days of antitoxin medical practitioners used their discretion and ingenuity in the treatment of diphtheria, but since von Behring showed the way, it would be regarded as irresponsible if antitoxin were not applied. The individual members of the medical profession need not be bacteriologists nor need they study the modern doctrines of immunity to be able to apply biological remedies to bacterial infections or poisoning by organic toxins. In these circumstances a considerable amount of space in this journal has been devoted to a symposium on snake bite by Dr. N. Hamilton Fairley, Dr. C. H. Kellaway and Miss Beryl Splatt. In the issue of June 8 the eighth and ninth articles of the series have been published. The whole series represents the most complete and authoritative dissertation on the subject in existence. We offer no apology for the large amount of reading matter; the evidence had to be given and each step in the investigations had to be explained. Otherwise the didactic value of the articles would have suffered. In summary it may be stated that the death dealing dose of the venom of each common venomous snake in Australia has been determined, that the yield of venom from a single bite of each snake has been ascertained, that the mechanism of the biting has been examined and depicted, that the chemistry of the poisoning has been explored, together with the time that elapses between the bite and death, that the efficacy of ligature, incision, chemical applications and excision has been evaluated and finally that the importance of antivenom treatment has been reviewed. The story is complete and there is no longer room for the astounding differences of opinion that have characterized some of the communications on the subject in the pages of this journal.

The first point of importance as far as the bitten person is concerned, is the species of snake that has bitten. Dr. Fairley has shown quite clearly that the death adder stands easily first in its deadly effects. The next on the list is the tiger snake; then come the copper-head and the brown snake. Dr. Fairley doubts whether the black snake ever kills an adult, although it may cause the death of a child of low body weight. From his descriptions and from his illustrations the identification of each species becomes a simple matter. His wonderfully clear pictures of dental wax impressions of the bite of the several snakes will be found very useful when the snake itself has not been seen or recognized. The second point of importance is the determination of the dose of venom that has been delivered by the snake. The appearance of the bites may yield some information on this matter. If the snake can be secured, it becomes a relatively easy matter to ascertain how much residual venom is held. If the species of snake be known and an estimate can be made of the manner in which the bite was inflicted, it should be possible for a medical practitioner to form a correct prognosis in the absence of treatment. It is not unknown that a practitioner has attributed recovery to his treatment when a fatal dose has not been injected.

It is now beyond question that ligature with or without incision will not save a life if a fatal dose of one of the more dangerous venoms has been injected. If the ligature be applied within sixty or one hundred seconds of the bite, the absorption of the venom may be delayed a little; if this is accompanied by excision of the soft tissues surrounding the bitten area, some good may result. But it is unwise to rely on these methods of treatment. Antivenoms to death adder and to tiger snake are being prepared at the Commonwealth Serum Laboratories and will be available in all parts of the Commonwealth. It should be possible to obtain a supply in time to save life, particularly if use be made of air services when the patient is placed some distance from a capital city. That the biological remedy is the only one on which reliance can be placed, must be emphasized over and over again. If antivenom is not employed in the treatment of the bite of a venomous snake, the practitioner in charge will have to show that it was not available. He can have no other excuse. The symposium that has appeared in the pages of this journal, will be regarded as the classical work on Australian snake bite for many years to come.

Current Comment.

IRON METABOLISM.

IN a recent issue attention was drawn to some work by Donaldson on hæmochromatosis and to its bearing on the question of iron metabolism. Hæmochromatosis is a rare condition and, while it is essential that its nature should be understood, the metabolism of iron is much more important. Iron plays a large part in the metabolism of the body. It is found in its largest quantities in hæmoglobin. It is also found in the liver and in the spleen, kidneys, adrenal glands and bone marrow. There is a great difference between iron as it exists in the hæmoglobin and as it is found in the liver. The iron in the liver is probably stored up for future use. The liver is the great storehouse of the body. A long investigation has recently been undertaken by Cyril J. Polson into the effects of intravenous injection of large doses of dialysed iron into rabbits.[1] While it may be stated at the outset that Polson does not add much to the knowledge of the metabolism of iron (he does not claim to do this), there is much of interest in his experiments and some useful deductions may be made. In his first paper he considers what he calls his short experiments and in his second paper he deals with his longer ones. In the short experiments the longest interval between the last dose of iron and the death of the animal was two weeks. In the longer experiments the interval lasted up to fourteen months. A 10% dilution of colloidal iron (dialysed iron of the British Drug Houses, Limited) was used and in order to avoid what Polson calls large doses, quantities of five to ten cubic centimetres were used for repeated dosage. It should be pointed out that five to ten cubic centimetres of colloidal iron will contain 0·5 to 1·0 gramme of iron. Thirty milligrammes of iron per kilogram of body weight constitute a fatal dose in rabbits and it is thus obvious that the doses given were many times the fatal dose. The animals did not die; some of them died when further doses were administered and some lived for fourteen months. The only possible conclusion is that soon after injection the iron was rendered inert. This undoubtedly is what occurred. Polson points out that rabbit's serum in a dilution of one in twenty destroys the colloidal state of iron *in vitro*, a copious flocculent precipitate being at once produced. He presumes that the same change would take place *in vivo*. The presumption is justified.

The principal results found by Polson on histological examination were embolism and phagocytosis. The precipitate is carried to the lungs and collects in these organs as emboli. He holds that the iron is subsequently removed from the lungs by phagocytes. He gives details of the various reagents used to determine the presence of iron in his sections and as an additional check he stained at the same time other sections known to be free of iron. The analysis of dried organs was carried out by Neumann's process with the incorporation of modifications suggested by Abderhalden and Pincussohn. He describes two kinds of phagocytes in the lungs. One consists of endothelial cells common to most organs, held by him to play a purely passive rôle in the phagocytosis of iron, and the other is of a special type called into action for the special removal of foreign material. These are "large spheroidal cells of similar size to the splenic phagocytes, but unlike them in appearance, the latter being oval or round." Although he admits that they may be wandering cells brought by the blood stream, he regards them as being of epithelial origin. He holds this view in spite of his observation that they are found in the lymphatics and he also points out that amœboid properties are exceptional attributes of epithelium. It was found in the early experiments that at the conclusion of the dosage the lungs contained 20% to 25% of the total iron administered. By the end of two months 16% was recovered from the lungs and thereafter from 4% to 5%. Polson thinks that the phagocytes are brought to the lungs by vessels not blocked with emboli and he postulates for these phagocytes the

[1] *The Journal of Pathology and Bacteriology*, July, 1928, and April, 1929.

power to pass through the endothelial walls of the blood vessels, to ingest the iron and to pass out again. He has not seen the phagocytes making this passage, but has seen them inside and outside the walls. It may be granted, however, that this process does occur, for it has been noted by others. While it cannot be denied that phagocytosis would occur if the phagocytes could gain access to isolated granules of iron, it must be clear that phagocytes would have great difficulty in absorbing granules from a glutinous mass of flocculent precipitate such as is described as being found in the lungs by Polson. Polson has ignored the quantity of iron which would be removed by solution or washing out. This has been proved to occur with nickel, a metal very closely allied to iron.

As far as the liver is concerned, Polson found that the Kupffer cells (in common with the phagocytes of the spleen) had the greatest avidity for iron. Although iron was found in the Kupffer cells from the commencement of the experiments, it did not enter the liver cells until the end of the second week after the injections. It is concluded that the iron reached the liver *viâ* the Kupffer cells and Polson holds that the iron is transferred to them from the lungs. No iron was found in the portal system. It was noticeable that there was a difference in the time taken for liver gain and lung loss; the maximum liver gain occurred at six to eight months and the maximum fall of iron content in the lung occurred at two months. The explanation offered by Polson to account for this difference in time is that the phagocytes are carried to the spleen when the pulmonary vessels become clear and are stored there for a time. He points out that the phagocytes of the lung and spleen are similar in appearance and that the maximum iron content of the spleen is coincident with the lowest point of iron content of the lungs. He draws attention to the fact that when iron is introduced into the body by any channel other than the veins, but little iron is found in the spleen.

In considering Polson's work the most important fact to be remembered is that he is dealing with entirely artificial conditions. As already pointed out, the iron injected into his animals tended immediately to become inert. It was very different from the organic albuminate of iron which comes from the food. His observations also serve to emphasize the known fact that it is exceedingly difficult to induce the absorption of iron and its utilization in the metabolic processes of the body. It is probable that when iron is given therapeutically, a small amount is absorbed; at the same time it is doubtful how far the iron thus absorbed is used in the formation of hæmoglobin. This has been shown in experiments on animals. Milk contains a low percentage of iron when compared with other foods. If a comparison is made of rabbits which have been allowed to change to a diet of green vegetables after a normal period of lactation, with those which have been brought up on an exclusively milk diet, the former are found to contain more hæmoglobin than the latter. If, however, inorganic salts of iron are added to the milk diet, the total hæmoglobin in the animals is not increased and they do not attain normal dimensions, although they grow much more rapidly than the similarly fed animals which did not receive iron. It is not by injection experiments that light will be thrown on the metabolism of iron.

ILEOSTOMY IN EXPERIMENTAL GENERAL PERITONITIS.

IN a report on some experiments on the treatment of acute general peritonitis in the dog, Thomas G. Orr and Russell L. Haden stated that enterostomy as an aid in the treatment of peritonitis has attracted the attention of clinicians during recent years.[1] They have operated on normal dogs and have produced a general peritonitis by freeing the appendix at the base and ligating it with tape. At the same operation they have divided the ileum fifteen centimetres above the ileo-caecal junction, invaginated the distal portion and brought the proximal portion to the surface through a wound and sutured it to the skin and fascia. In one series of nine animals the length of life varied from two to six days, with an average of a little over three days. The findings on chemical analysis of the blood were similar to those observed in general peritonitis, namely, an increase in non-protein and urea nitrogen, a decrease in the chlorides and no constant change in the carbon dioxide combining power. In a second series of six animals the same operation was performed, but the animals were treated with forty cubic centimetres of 1% sodium chloride per kilogram of body weight. These dogs lived from four to eighteen days, an average of ten and a third days. The changes in the blood chemistry were not pronounced; in some instances there was a terminal rise in the non-protein and urea nitrogen and in one instance there was a rise in the chlorides. The conclusion is that in experimentally produced general peritonitis drainage of the gut by ileostomy has no beneficial effect.

At the time when the ileostomy was not performed, the dogs were not suffering from peritonitis. The ileostomy was carried out at the same time as the ligature of the appendix. When ileostomy is performed in general peritonitis, ileus is present and the object of the ileostomy is to overcome the obstruction until such time as the paralytic condition of the bowel has disappeared. In the dogs experimented on there is no hope that the ligated terminal part of the bowel will resume its functioning. Although Orr and Haden do not apply their findings to peritonitis in human beings, their opening sentence quoted above suggests that their work may be considered as bearing on the condition. Enterostomy or entero-colostomy advocated by Handley is a desperate measure used as a last hope and is sometimes effective.

[1] *The Journal of Experimental Medicine*, April, 1929.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Relation of Heart Beat to Skeletal Muscle Tone.

WHENEVER the knee jerk is recorded as vertical lines on a slowly moving kymograph, there are scarcely to be found two consecutive reactions of the same magnitude. F. E. Emery (*American Journal of Physiology*, April, 1929) has examined the effect of the phases of the heart beat on the knee jerk, arguing that the systolic discharge increases the pressure throughout the arterial system and increases slightly the turgor of the muscles. Simultaneous records were taken of the carotid pulse and the knee jerk on a rapidly revolving kymograph. A careful examination of some hundreds of reactions shows that the greatest height of knee jerk occurred during the systolic phase of the heart's action. The data show that, whatever the causative factor or factors may be, there is present more tonus in the skeletal muscles during the systole than during the diastole of the heart.

Changes in Striped Muscle After Sympathectomy.

B. E. GAISSINSKY AND M. I. LEWANTOWSKY (*Journal of Physiology*, February, 1929) have studied the changes in striped muscle subsequent to the destruction of the sympathetic nervous supply to a limb. Dogs were subjected to ramisection from the first lumbar to the third sacral segment on the left side. Portions of muscle were removed four months later from corresponding points in the flexor and extensor muscles of both hind limbs. Sections revealed progressive loss of transverse and longitudinal striation, hyalinization and an increase of connective tissue. The connective tissue and fat deposits were increased, especially near larger vessels. The processes were more advanced in the animals in which the removal of the sympathetic was the more extensive. The histological changes were degenerative and did not seem to depend upon changes in the vascular supply. After ramisection the blood returning from the limb deprived of its sympathetic nerve supply manifested a decrease of calcium and chlorine and no change or a slight increase in potassium and phosphate.

Acetylene for the Determination of Cardiac Output.

VARIOUS methods have been described during the past few years having for their object the determination of cardiac output. Several of these methods are based on the measuring of the rate with which a gas like nitrogen, nitrous oxide or ethyl iodide is exchanged during the passage of blood through the lungs. Various objections can be raised to all these methods. A. Grollman (*American Journal of Physiology*, April, 1929) has devised a method, using acetylene as the inert gas. He shows that acetylene is much more ideal than ethylene, nitrous oxide or ethyl iodide, a superiority resting on its increased solubility over ethylene or nitrous oxide in blood, the constancy of this solubility as compared with these gases or ethyl iodide and the ease with which analyses and other manipulations can be carried out with acetylene as compared to any of the other gases. The method is quite simple. The subject breathes from a bag containing about 8% to 10% of acetylene in air enriched with oxygen. The mixture is rebreathed for a time sufficiently long to bring about mixing in the lung-bag system and a sample is taken and analysed. After another five seconds' rebreathing another sample is taken and analysed. By a simple calculation the cardiac output is estimated from the data given by the analyses. The estimation of acetylene is simple, the gas being absorbed in an alkaline solution of mercuric cyanide. Consecutive determinations on the same individual showed a variation of about 2% in the values of the cardiac output.

Sympathetic Innervation of Skeletal Muscle.

G. T. POPA AND F. POPA (*Journal of Physiology*, February, 1929) have examined the function and morphology of the sympathetic innervation of the skeletal muscle in the wing of the pigeon. From one to five thoracic ganglia immediately below the cervical ganglion were removed on one or both sides. The results confirm those of John Hunter and are mainly: (i) Immediate, in addition to changes in the eye and feathers, there is a definite drooping of the wing; (ii) remote, sensibility to cold and appetite increased. Even after two months when the birds are active and lively, the wing from which the sympathetic has been removed, is easily fatigued. Effects are always more pronounced as the number of ganglia removed is increased. Excitation of the sympathetic cord between spinal nerves 11 to 14 invariably produced quick contraction of the wing. Excitation of the dorsal aspect of the sympathetic cord produced depression of the neck feathers and contraction of the dorsal muscles of the neck. Excitation of the ventral aspect of the same part of the cord gave contraction of the wing and ventral muscles of the neck. This indicates a topographic localization in the trunk of the sympathetic. The brachial plexus and the sympathetic were exposed and the sympathetic stimulated for five to ten minutes, blood was collected from the aorta and kept in citrate solution for twenty-four hours in the ice chest. Blood similarly treated from a normal bird was used as a control. The first serum injected into pigeons caused liveliness, ruffled feathers and high position of the wings, with occasional extension of the tail. The power of contraction of the wing was definitely increased. The normal serum caused but slight increase in power. Blood collected three hours after decerebration and injected into normal birds produced high position of wing, general excitement and "clasp knife" contraction.

Pneumin, a Respiratory Autocoid.

SWALE VINCENT AND J. H. THOMPSON (*Journal of Physiology*, February, 1929) bring evidence to show that the adrenal cortex secretes a substance called by them pneumin, essential for the normal movements of respiration, which passes through neighbouring lymphatic glands and into the veins by way of the thoracic duct and right lymphatic duct. Using decerebrate cats which normally would breathe for several hours, they found that after extirpation of the adrenal cortex on both sides there was irregularity, intermittence and finally cessation of respiratory movements within half an hour. With artificial respiration the animal could be kept alive indefinitely. Ligaturing the total blood supply to both adrenals has the same effect (somewhat delayed) as total extirpation, although ligaturing the veins has no effect. Damage to the lymph drainage of the adrenals has the same effect. Ligature of the large veins in the neck below the entrance of the lymphatic ducts has the same effect as removal of the adrenals. Something seems then to escape from the adrenals into the lymphatics which is essential to normal respiration in the cat. Intravenous injection of fresh adrenal extract when respiration has ceased completely restores normal breathing in two or three minutes. Injections of adrenalin at this stage have no effect.

Consecutive Fractional Tests in Gastric Analysis.

WHILE the fractional method of gastric analysis is very greatly used, there is very little information available as to possible daily variations in the results obtained from the same individuals. I. E. M'Cracken (*Edinburgh Medical Journal*, December, 1928) has made an extensive study by consecutive tests of seventy-five persons presenting gastric symptoms. The ease with which the tube is swallowed, makes for considerable differences in the response on different occasions on the same individual. About 62% may be expected to swallow the tube easily on the first attempt. On the occasion of a subsequent test 87% may be expected to swallow the tube easily. When attempts are made to classify the acid curves according to an existing nomenclature, the type of curves obtained by consecutive tests at short intervals of time was found to differ in about 47% of the cases. The curves from consecutive tests approximate most nearly to one another at a period of one and a half hours after the commencement of the test. In the majority of cases the first test yielded a higher response than a second test. This was true at all the quarter of

an hour intervals of the test except one hour after the commencement of the test, at which period a greater response was on the average obtained from the second test. When patients who experienced difficulty in swallowing the tube at the first test, are considered in a group by themselves, it was found that the majority of such patients showed a greater response from the second test, at which time the tube was usually swallowed with greater ease. The variations obtained in different consecutive tests were greater than is commonly supposed. It is probable that the average amount of variation observed is insufficient to affect seriously the diagnostic significance of the results obtained from this test. In certain cases more extreme variations do occur and in these the interpretation of the results must be to some extent erroneous. It is evident that fractional analysis cannot be considered a reliable method of recording change, the result of treatment or other cause, in the gastric function of an individual.

BIOLOGICAL CHEMISTRY.

Blood Chemistry in Menstruation.

DURING an investigation into certain forms of Bright's disease H. G. Close and A. A. Osman (*Biochemical Journal*, Volume XXII, Number 6) sought to determine the possible variations in some of the constituents of the blood which might occur during the menstrual cycle. The determinations were made on thirteen normal, healthy young women and corresponding determinations were made on women of a different social class who, though not diseased, were in a state of subnormal health due to living under adverse circumstances. The results from this group will be published later. The blood was collected at midday under paraffin from a vein without exertion and determinations made within a few hours. Observations were usually made midway between menstrual periods on the first, second or third day and covered at least two complete menstrual cycles. The serum carbonate, serum chloride, inorganic phosphate, calcium and urea were estimated and the methods used are given. The tables which accompany this article, reveal no significant increase or decrease in the blood calcium or blood urea, a slight but definite increase in the serum chlorides during the flow and to some extent a slight decrease in the serum carbonate. No change was observed in the inorganic phosphate. There was no significant difference in the figures obtained on the first, second or third days of the menstrual flow.

Glucose in Normal Urine.

ALI HASSAM (*Biochemical Journal*, Volume XXII, Number 5) has recorded certain observations made while applying the osazone test to a number of specimens of urine from healthy Egyptian students and has confirmed the older view that glucose may be a constituent of normal urine. Charcoal was used to remove the interfering substances present in normal urine and clear, well crystallized osazones were obtained with the usual phenylhydrazine hydrochloride and sodium acetate mixture, twelve to fifteen hours being allowed for the crystals to form. With this technique typical glucosazone crystals were obtained from aqueous solutions of glucose containing 2·5 milligrammes per cubic centimetre. Normal urine gave numerous types of crystal mixtures which were shown to consist of two simple osazones, one identical with glucosazone, the other corresponding with Baisch's iso-maltosazone as regards melting point, but differing in crystalline appearance. The urine from over 700 persons was examined under different conditions and typical glucosazone crystals were given in 20% to 30% of the specimens voided one to two hours after any ordinary meal and fell to 7% after a twelve hours' fast. Glucose tolerance tests were done on many of the students and no abnormality of carbohydrate metabolism was found. Hassam also shows that some of Höst's physiological osazones are impure crystal mixtures and suggests that the same applies to some of Geelmuyden's osazones.

Blood Sugar.

O. J. NIELSON (*Biochemical Journal*, Volume XXII, Number 6) reviews the literature and has undertaken an investigation into the oscillations of blood sugar values as distinct from the regular curve waves. The blood sugar values were determined by the Hagedorn-Jensen method and the urinary sugar values were determined qualitatively by Fehling's and Almen's method and quantitatively by fermentation or by the Benedict-Osterberg method for total sugar determination. All the patients had been perfectly quiet in bed for ten to fifteen minutes before the first sample of blood was taken and throughout the period of blood-taking, so that the results were not influenced by muscular work. The determinations were made on fasting individuals at intervals of one to five minutes for a period of one and a half to two hours. In a group of five patients with perfectly normal carbohydrate metabolism the curve was almost horizontal without any oscillations, as the variations in blood sugar do not exceed the limits of experimental error. In diet-treated diabetics in every instance it was found that at perfect rest the high fasting blood sugar value decreased gradually and smoothly without any oscillations in the curve during the two hours' observation. In one diabetic patient not treated with "Insulin," whose diabetes was complicated by nephritis, there was practically no fall in the increased blood sugar during seventy minutes of observation and there was no oscillation in the curve. In diabetics treated with "Insulin" he found two different forms of blood sugar curve: one, the most frequent, in which the blood sugar rises during fasting; the other, more rare, in which the blood sugar falls during fasting. No oscillation was noticed in any of the patients examined. In diabetics at different times of the day after ingestion of food or administration of "Insulin" no oscillations were found, there were only the well known rises and falls and waves in connexion with the "Insulin" and food intake. Blood sugar curves after glucose on patients with normal carbohydrate metabolism show no oscillations. Blood sugar curves on various days in patients on a mixed diet did not give uniform results, the curves differing considerably in the time of rise and in the waves on different days. In regard to the elimination of the sugar by the urine, however, about the same total amount of glucose was eliminated on each day of experiment. In this group the patients were given a 10% watery solution of one gramme of glucose per kilogram of body weight and rested quietly in bed during the experiment. In one instance only did the investigator succeed in obtaining uniform curves after ingestion of glucose on three different days and this was in a patient who throughout the experimental period received every day the same amounts of carbohydrate, protein, fats, salts and liquids and whose carbohydrate metabolism was not quite normal.

Urea Concentration Test.

G. A. HARRISON (*The Lancet*, November 24, 1928) stresses the fact that tests of renal efficiency are relatively gross and conclusions drawn from results apply only on the day of the test, also that investigations must be repeated at intervals as guides to prognosis and treatment. In regard to the technique of the test, he allows the patient to have a dry breakfast on the morning of the test, but nothing to drink after 10 p.m. the previous night. Occasionally, in spite of withholding fluids for eight to twelve hours previously, there may be an excessive diuresis owing to the release of water previously retained in the tissues. It is merely a matter of convenience whether the test be repeated on the same or another day, there being no objection to giving a second dose of urea two or three hours after the first. The doses of urea found satisfactory for children are given in detail. He also mentions the importance of measuring the volume of each specimen of urine and states that if the volumes are larger than one hundred and twenty cubic centimetres in the first hour or one hundred cubic centimetres in the second and subsequent hours, a low concentration of urea does not necessarily indicate deficient renal function. In judging the efficiency of the kidney it is of no importance whether the maximum concentration of urea occurs in the first, second or third hour. Figures between 2% and 2·5% may be regarded as on the boundary zone; below 2% they indicate renal inadequacy, provided that the test has been properly executed.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Children's Hospital, Adelaide, on February 28, 1929. The meeting took the form of a series of clinical demonstrations.

Pernicious Anæmia.

DR. A. R. SOUTHWOOD showed two patients who were suffering from pernicious anæmia, to illustrate the blood changes following liver feeding. The immature red cells (reticulocytes) had been counted daily and their number had been expressed as a percentage of the total red cell count; on the fifth day of liver feeding the reticulocyte count had begun to rise and by the twelfth day the reticulocytes had comprised 26% of the red cells. A rapid descent had then followed and by the thirtieth day the reticulocytes had fallen to the normal level of 1%. During this period the red cell count had gradually risen, in one instance passing from 1,500,000 per cubic millimetre to 4,500,000 in seven weeks. Hydrochloric acid had been administered simultaneously. Patients who had been given liver but no hydrochloric acid, manifested less rapid improvement. The mode of action of liver feeding was discussed and it was shown that subcutaneous injection of a suitably prepared liver extract (carried out by Dr. E. McLaughlin, of the Adelaide Hospital Laboratory) produced similar blood changes to those obtained from oral administration. The increased reticulocyte percentage was the first definite indication of the effectiveness of the liver diet in any particular patient.

Parkinsonism.

Dr. Southwood also showed a patient suffering from Parkinsonism, probably postencephalitic in nature. The leading features of the syndrome were demonstrated. Hyoscine administration had effected partial improvement. On the suggestion of Dr. F. S. Hone it was proposed to treat the patient with large doses of tincture of stramonium which had been found useful by recent investigators.

Ringworm.

DR. W. UPTON showed a patient with extensive ringworm of the scalp. The patient was a female, aged seven years, who gave a history of areas of partial baldness, accompanied by a scaly condition of several months' duration. These lesions had been treated with various antiseptic ointments, but the condition was spreading. When ultraviolet light was applied to the affected areas in a darkened room and a piece of "Wood's glass" was interposed, a brilliant fluorescence occurred. Some of the affected hairs gave the appearance of having been coated with an aluminium paint. This appearance was regarded as diagnostic. In addition a microscopical examination of these hairs showed that they were crowded with "ringworm spores."

It was intended to admit the child to hospital for thallium acetate epilation therapy.

Epilepsy.

DR. R. THOROLD GRANT showed a girl, *ætatis* eleven years, who had suffered from epilepsy for years, having had as many as a hundred fits a day on one occasion. The fits had been controlled by the administration of "Luminal" and bromide and later by the use of Gelineau's *dragées*. She had recently developed a large left-sided pleural effusion. There was a history of contact with a tuberculous relative. The Mantoux tuberculin test yielded a positive reaction. Aspiration of the fluid had resulted in a pneumothorax with complete collapse of the left lung. The classical signs of hydro-pneumothorax were now present. During the early stages of this latter illness all sedative medication had been stopped and the child had not had a fit for three weeks; this was the longest period for which she had been free for many years.

Hemichorea.

Dr. Grant also showed a girl, *ætatis* nine years, who suffered from hemichorea. He pointed out that definite hypotonus was present.

Polycystic Disease of the Kidney.

DR. OWEN M. MOULDEN showed a female patient, aged fifty-five years, who had been first seen on June 25, 1928. She had complained of dyspnœa on exertion of a slight degree and of throbbing in the neck for four or five years. She had also complained of bilateral backache just below the shoulder blades; otherwise she felt well except for general lassitude. She had been told that she had a floating kidney some years previously.

On examination the pulse rate had been 90. The apex beat had been in the nipple line and the heart sounds normal except for an accentuation of the second sound at the base. The systolic blood pressure had been 190 millimetres of mercury and the diastolic pressure 110 millimetres. The specific gravity of the urine had been 1006 and a moderate cloud of albumin had been present. On palpation of the abdomen a large mass had been felt filling each flank. The masses were hard; fixed and large rounded bosses could be felt over the surface of each mass. Cystoscopy with catheterization of the ureters had revealed a normal bladder with normal flow of urine of normal appearance from each ureteric orifice. The urine from each side had been normal microscopically and on culture.

The urea percentage in the urine from the right side had been 0·6 and that from the left 0·4 at the end of the second hour with McLean's technique. The blood urea had amounted to 62·5 milligrammes per 100 cubic centimetres.

X ray examination had revealed great enlargement of each kidney, the lower pole on each side occupying the iliac fossæ. Pyelography had revealed a definite enlargement of each renal pelvis to about three times the normal size with filling defects due apparently to the intrusion of rounded masses into the lumen of the pelvis and calyces on each side.

For three months following the examination the patient had rested, had reduced her food intake and emptied her bowels daily with a saline aperient. The daily excretion of urine had varied between about two and two and a half litres (sixty-five and eighty ounces) during this period.

A further examination had then revealed urine of a specific gravity of 1002, containing a slight cloud of albumin. The urea concentration test (McLean) had yielded figures of 0·7%, 0·6% and 1·0% at the end of one, two and three hours respectively. The blood urea at the time of the meeting was 25 milligrammes per 100 cubic centimetres. The patient had remained in comparatively good health and had lost no weight.

Apart from the physical findings, the point of interest was the effect of rest and diminution of food intake on the renal functions. She appeared to have well compensated kidneys, for although they possessed only about half the normal power of urea excretion they were excreting about double the normal amount of urine.

Œsophageal Diverticulum.

DR. H. M. JAY showed a woman, aged thirty-eight years, who was suffering from an œsophageal diverticulum. She had reported first in October, 1925, complaining of husky voice and incessant cough which had worried her for two or three years previously and had been diagnosed as hysterical by those medical men who had seen her. He had been unable to see any morbid changes in the upper air passages and there had been no abnormal signs detected in the chest. The cough had been of the "irritable" type and unaccompanied by expectoration. In consultation with Dr. Lendon it had been agreed that the condition was similar to that sometimes seen in myxœdema and, as the patient's appearance and other symptoms and signs suggested this affection, she had been given a course of thyreoid extract. This, however, had not improved her. The possibility of the presence of an œsophageal pouch pressing on the trachea or causing irritation of the recurrent laryngeal nerve had then

occurred to him so at his request Dr. Nott had taken a series of X ray pictures which clearly demonstrated the presence of a diverticulum in the middle third of the œsophagus.

Dr. Jay said that the ætiology remained obscure, though it seemed almost certain to him that the pouching was due to traction and not to pulsion. The patient was in good health, her blood had given no reaction to the Wassermann test and the length of the history excluded the presence of any malignant growth. Only recently had there been any discomfort following the taking of food.

Sequel to Mastoid Operation.

Dr. Jay's second patient was one on whom he had performed a Heath's mastoid operation three years previously. He was exhibiting her to show a rather unusual sequel. As frequently happened, the mastoid antrum had become closed off by an epidermal curtain, but there was so free a communication with the naso-pharynx through the antrum, *aditus*, middle ear and tube that this structure moved in and out with inspiration and expiration. This seemed to him to provide a good demonstration of the functional result obtained by the operation.

Skiagrams.

DR. H. A. MCCOY showed a series of skiagrams.

Two examples of duodenal diverticulum were illustrated and in one a single diverticulum was present in the second stage of the duodenum. In the other two diverticula were demonstrable, one in the second stage and the other one, a large pedunculated pouch, was seen arising from the third stage. It filled the space enclosed by the duodenal loop overlying the head of the pancreas.

A cyst in the right lobe of the liver was demonstrated. In the wall of the cyst a large amount of calcium salts was present.

Films were shown of a child, aged four weeks, in which the abnormality of the lower portion of the right leg and foot was found to be due to complete absence of the fibula and of the centres for the *talus*, cuboid and the two digits on the fibular side of the foot. There was in addition delayed development in the upper epiphysis of the tibia and in the centre for the *calcaneus*.

DR. H. C. NOTT showed a series of skiagrams demonstrating various developmental anomalies and pathological conditions of the ureters and renal pelves as shown by opaque injections; also some artificial pneumo-ventrilograms produced by the introduction of air through a lumbar puncture.

Fracture of Vertebræ.

DR. D. R. W. COWAN and DR. L. O. BETTS reported a case of fracture of the vertebræ. This report will be published in a subsequent issue.

Tumour of the Femur.

DR. E. BRITTEN JONES showed a bone tumour involving the lower fifteen centimetres (six inches) of the shaft of the left femur. The patient was a male, aged thirty-five years, who stated that he had knocked his left knee early in January, 1929. Since then he had suffered with a nagging pain, worse at night, situated just above the knee. Five years ago he had suffered from a discharging sore just below the left knee. It cleared up after five weeks' treatment. On examination of the left leg a "tissue paper" scar was present on the inner aspect of the left leg just below the knee joint; there was no swelling of the limb. On palpation a bony tumour was found to be present which involved the outer aspect of the lower fifteen centimetres (six inches) of the shaft of the left femur.

The Wassermann test yielded a strongly positive response. A radiographic report stated that there was new periosteal bone formation involving the outer aspect of the lower femoral shaft—probably an early sarcoma.

Dr. Britten Jones proposed treating the patient with anti-syphilitic remedies. Further radiograms would be taken and the response or otherwise to treatment would be noted.

Rupture of the Biceps Brachii.

Dr. E. Britten Jones also demonstrated a traumatic rupture of the long head of the *biceps brachii* in a male, aged fifty-three years. The patient's hook had slipped whilst he was moving a bale of wool and he had struck his left arm against an iron stanchion. His forearm had been flexed and all the muscles taut at the moment of impact. He had complained of a sudden sharp pain in the upper third of his left arm and had stated that his whole arm felt useless. He had attended the out-patients' department of the Adelaide Hospital four days after the accident. On examination there had been a distinct gap in the upper limit of the fleshy portion of the biceps muscles on the left side. No bruising of the tissues had been present.

On palpation with the muscle contracted, separation of the fleshy fibres from the tendon of the long head was seen to be present.

In reply to a question Dr. Britten Jones said that he intended to suture the tendon to the fleshy belly of the muscle.

Cinematography in Nervous Conditions.

Dr. E. Britten Jones showed a cinematograph film of a father and daughter affected by arsenical polyneuritis and hereditary ataxia respectively. The father had taken a large single dose of an arsenical compound with suicidal intent and four days later he had noticed difficulty in doing up his buttons *et cetera*. The ataxia had gradually increased and had reached a maximum in a fortnight. He had presented the syndrome of arsenical pseudotabes.

The daughter, aged eleven, had been apparently normal until the age of four, when it had been noticed that she gradually became unsteady in walking and that she tended frequently to fall over. In the course of a year or two the unsteadiness had spread to the upper extremities and she had been unable to lift a full cup without spilling its contents. Her paternal uncle had had a similar unsteadiness and had died at the age of twenty-six.

Clinically her condition resembled Friedreich's ataxia in that *pes cavus* and scoliosis were present with absence of the patellar reflexes. However, the plantar response was not definitely extensor, nor was nystagmus present; the ataxia was progressing slowly.

Dr. Britten Jones thought that the condition should be classed as one of Friedreich's ataxia. He thanked Dr. Moulden for assisting him in taking the pictures.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Bohrsman, Otto, M.B., M.S., 1893 (Univ. Aberdeen), 185, Elizabeth Street, Sydney.

Lorger, Albert Eric, M.B., Ch.M., 1926 (Univ. Sydney), Parkes, New South Wales.

Medical Societies.

THE MEDICAL WOMEN'S SOCIETY OF NEW SOUTH WALES.

A MEETING OF THE MEDICAL WOMEN'S SOCIETY OF NEW SOUTH WALES will be held on Friday, June 21, 1929, at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, at 8 o'clock p.m. DR. MARGARET HARPER will give an address on her experiences abroad.

All medical women registered in New South Wales are invited to attend this meeting. Those desirous of joining the Society are requested to communicate with the Honorary Secretary, DR. M. M. HAMILTON, 93, Macquarie Street, Sydney.

Correspondence.

SENSITIZATION TO MILK.

SIR: My domestic milk supply comes from our own farm and I am able to observe the conditions under which it is produced. Each year when green maize or maize ensilage was fed to the cows my little daughter suffered from vomiting and diarrhœa. This sensitive reaction to maize-fed milk lasted up to the eighth year of her age. When her condition indicated that the maize feeding season had begun, it became necessary to use the milk from a cow from which maize was withheld. There was no doubt in my mind of the coincidence of cause and effect.

I record this observation as it is possible that it bears some relation to the ætiology of "summer diarrhœa." One can well imagine the disastrous consequences of persisting in feeding maize-fed milk to an infant sensitive to its effects.

Yours, etc.,

JOSEPH T. HOLLOW,
Medical Superintendent.

Mental Hospital, Kew,
Victoria,
May 18, 1929.

Obituary.

DUNCAN GLENORCHIE ROBERTSON.

WE regret to announce the death of Dr. Duncan Glenorchie Robertson which occurred at Perth on June 9, 1929.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

30-34, Elizabeth Street,
Sydney,
New South Wales,
June 5, 1929.

The Editor,

THE MEDICAL JOURNAL OF AUSTRALIA,
Seamer Street, Glebe.

DEAR MR. EDITOR,

I have been requested to write a few notes about the forthcoming Congress of the Australasian Branches of the British Medical Association, so, first, I take this opportunity to express, through THE MEDICAL JOURNAL OF AUSTRALIA, my appreciation of the very great honour which the members of our profession have conferred upon me in appointing me to the exalted position of President, and I desire to assure them that I shall do my best to assist in making our Congress a great success.

The various committees are working with great enthusiasm and the attendance at all their meetings is very satisfactory. The members of the medical profession in this State are becoming deeply interested in the business of the Congress and prospective visitors from the other Australian States and from New Zealand are beginning to look forward to a pleasant and instructive meeting.

We are all delighted to hear that we shall have, as distinguished visitors from Great Britain, Sir Ewen Maclean, President of the British Medical Association, and Professor Hugh Maclean, of St. Thomas's Hospital.

I wish to appeal to the profession in New South Wales to do all that is possible to make the event worthy of this great Mother State of Australia and to our professional brothers and sisters in the other States and in New Zealand to assist us by their presence and I can assure our visitors that we shall extend to them a very hearty welcome and shall do all in our power to make their visit happy and enjoyable.

In conclusion, I should like to express to you my appreciation of the very great assistance that is being afforded to Congress by THE MEDICAL JOURNAL OF AUSTRALIA.

I remain, dear Mr. Editor,

Faithfully yours,
G. H. ABBOTT,
President.

THE work of the honorary secretaries of the sections of the third session of the Australasian Medical Congress (British Medical Association), Sydney, 1929, is now far advanced. Those who are desirous of reading papers before the various sections, should lose no time in making application. It is assumed that all papers are nearly ready for submission.

Section of Neurology and Psychiatry.

Provisional arrangements have been made for the holding of combined meetings of the Section of Neurology and Psychiatry and other sections. The programme will include a combined meeting with the Sections of Medicine, of Surgery and of Orthopædics at which trauma in relation to functional nervous disorders will be discussed. The speakers will be Dr. W. M. Macdonald, of Dunedin, Professor J. P. Lowson, of Brisbane, and Dr. John Bostock, of Brisbane. A second combined meeting will be with the Section of Ophthalmology, at which the value of ocular signs in neurological diagnosis will be the subject of discussion. Dr. A. W. Campbell, of Sydney, will be the chief speaker of the neurological side.

The subject of the pyrexial treatment of nervous and mental disorders, with special reference to the treatment of general paralysis of the insane by malarial infection, will be opened by Dr. Clifford Henry, of Sydney, Dr. Reginald Ellery, of Mont Park, Victoria, and Dr. John Bostock, of Brisbane. A bioscope film depicting patients undergoing treatment by the injection of blood containing the plasmodium of malaria will be exhibited.

One morning will be devoted to a discussion on mental hygiene and on the proposed formation of a national council of mental hygiene. Among the other papers to be read will be one on the psycho-galvanometer by Professor W. S. Dawson, of Sydney, one on confusional psychoses by Dr. S. Evan Jones, of Broughton Hall, New South Wales, and one on the educational aspect of stammering by Dr. T. Garnet Leary, of Melbourne.

A clinical meeting will be held at Broughton Hall Psychiatric Clinic; a visit will be organized to the Glenfield Special School for Mentally Defective Children; a demonstration of pathological specimens will be arranged by Dr. Oliver Latham at the Medical School, University of Sydney.

The joint honorary secretaries of the section are Dr. J. A. L. Wallace, Callan Park, New South Wales, and Dr. R. A. Noble, 143, Macquarie Street, Sydney.

Section of Dermatology.

Dr. R. R. Wettenhall, of Melbourne, has accepted the position of President of the Section of Dermatology. He will visit Sydney in a short time for the purpose of discussing the arrangements of the section with the Honorary Secretary, Dr. George R. Hamilton, 147, Macquarie Street, Sydney. Dermatologists who propose to join Congress and to attend the meetings of this section, should communicate with Dr. Hamilton as soon as possible.

Dr. J. H. Kelly, of Melbourne, will read a paper on the gold treatment of lupus erythematosis and Dr. R. C. E. Brodie, of Melbourne, will read a paper on Bucky's *Grenzstrahlen* (rays at the extreme ends of the spectrum) in the treatment of dermatological conditions.

Accommodation.

As the accommodation in hotels in Sydney and the immediate suburbs is limited, members who intend to be

present at the third session of Congress, are advised to secure rooms at an early date. Application should be made to the manager of the hotel selected. Should any member have any difficulty in securing suitable accommodation, he is advised to communicate with Dr. R. A. R. Green, 175, Macquarie Street, Sydney, the Honorary Secretary of the Hotels and Lodgings Committee. The accompanying list will be found useful.

HOTELS IN SYDNEY.

Hotel.	Address.	Tariff.	
		Per Day.	Per Week.
Aaron's Exchange	Gresham Street	10s. 6d.[1]	—
Adams's (Tattersall's)	259, Pitt Street	20s. to 22s.	£6 2s. 6d. to £7.
Arcadia	Pitt Street	15s. 6d.	—
Australia	Castlereagh Street	From 15s.	—
Carlton	56, Castlereagh Street	22s. 6d.	—
		14s.[1]	
Grand Central	Clarence Street	5s. to 7s. 6d.[2]	—
Métropole	Bent Street	From 19s	—
		From 7s.[3]	
Petty's	York Street	From 22s.	—
		14s. 6d.[1]	
Sydney	Opposite Central Station	13s. to 14s.	£4 3s. to £4 9s. 6d.
		19s. 6d. to 20s. 6d.[4]	£6 3s. 6d. to £6 9s. 6d.
Usher's (Metropolitan)	64, Castlereagh Street	From 25s.	—
Wentworth	Church Hill	From 27s. 6d.	—
Windsor	Park and Castlereagh Streets	From 17s	£5 5s.

BOARDING HOUSES AND SUBURBAN HOTELS.

Hotel or Boarding House.	Address.	Tariff	
		Per Day.	Per Week.
Belvedere	81, King's Cross Road, Darlinghurst	From 12s. 6d.	From £3 13s. 6d.
Bondi	Bondi Beach	—	£6 6s.
Chevereil's	Elizabeth Bay Road	—	£5 5s.[5]
			From £10 10s. to £14 14
Clifton Gardens Hotel	Mosman	—	From £5 5s.
Coogee Bay	Arden Street, Coogee	From 20s.	From £6 6s.
Hotel Mansions	Bayswater Road, Darlinghurst	16s 6d.	From £5 5s.
International	Bondi Beach	From 21s.	From £5 5s.
Labrador	Macquarie Street	From 16s.	From £4 14s. 6d.
"52, Macleay Street"	Greenknow Avenue, Potts Point	—	£5 5s.[5]
			£10 10s.[7]
			£11 11s.[8]
			From £16 16s.[6]
Manly Hotel	Opposite Manly Wharf	From 21s.	£6 6s.
New Brighton	The Corso, Manly	—	£3 3s.[1]
			£5 5s.[4]
New Oriental Private Hotel	King's Cross	11s. 6d. to 13s. 6d.	£3 3s. to £4 4s.
Oceanic	Arden Street, Coogee	From 21s.	From £6 6s.
Pacific	Ocean Beach, Manly	—	Terms on application
Steyne	The Corso, Manly	—	£7 7s.
Tudor House	127, Phillip Street	14s.	£4 4s.
Y M C A Hostel	325 to 327, Pitt Street	6s. 6d.[1]	—
Y.W.C.A. Hostel	189, Liverpool Street	4s.[1]	—

[1] Bed and breakfast. [2] Bed only. [3] Single rooms [7] Double rooms.
[4] Meals as taken. [5] Inclusive. [6] Suites for two people. [8] Double rooms with bath

Post-Graduate Work.

POST-GRADUATE COURSES IN MELBOURNE.

THE Melbourne Permanent Committee for Post-Graduate Work has now fixed the dates for the six lectures to be delivered by Professor Hugh Maclean, of Saint Thomas's Hospital, London, as announced in our issue of April 27, 1929, page 610. The lectures will be delivered at the Medical Society Hall, Albert Street, East Melbourne, at 8.30 p.m. on each of the dates mentioned.

August 12, 1929.—"Modern Views on Gastric Physiology and Pathology."

August 14, 1929.—"Diagnosis and Treatment of Gastric Diseases."

August 16, 1929.—"Diagnosis and Treatment of Gastric Diseases."

August 19, 1929.—"Some Problems in Carbohydrate Metabolism and Diabetes."

August 21, 1929.—"Observations on Vascular and Renal Disease."

August 23, 1929.—"The Uses and Abuses of Biochemical Methods in Clinical Medicine."

The fee for the course is five guineas. Those who propose to attend, should send in their names and the fee to the Honorary Secretaries, Dr. J. W. Dunbar Hooper and Dr. H. R. Dew, 12, Collins Street, Melbourne, before the commencement of the course.

The annual refresher course of post-graduate lectures will be held during the fortnight from August 12 to August 23, 1929. This course will therefore coincide with the course of lectures to be delivered by Professor Hugh Maclean. The fee for the annual course is three guineas. Applications should be made for further details to the Honorary Secretaries. Arrangements can be made for a certain number of members to take up residence in some of the metropolitan hospitals during the period of the course. An additional fee for board will be charged.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned has been registered under the provisions of *The Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Davis, Harold Julian, M.B., B.S., 1927 (Univ. Adelaide), Launceston.

Books Received.

CONSTITUTIONAL INADEQUACIES: AN INTRODUCTION TO THE STUDY OF ABNORMAL CONSTITUTIONS, by Nicola Pende, M.D., Translated by Sante Naccarati, M.D., Sc.D., Ph.D., with a Foreword by George Draper, M.D.; 1929. Royal 8vo., pp. 270, with illustrations. Price: $3.50 net.

THE PRACTICAL MEDICINE SERIES: General Therapeutics, by Bernard Fantus, M.S., M.D.; Series 1928. Chicago: The Year Book Publishers. Crown 8vo., pp. 470, with illustrations. Price: $2.25 net.

MALARIA: LA VALEUR DE LA QUININE ET DU MERCURE DANS SON TRAITEMENT ET DANS LA LUTTE CONTRE LE PALUDISME, by H. E. Driessen; Preface de Jacques Peroni; 1929. Rome: Paolo Cremonese. Royal 8vo., pp. 192, with illustrations. Price: Frs. 35.

THE MEDICAL ANNUAL: A YEAR BOOK OF TREATMENT AND PRACTITIONER'S INDEX, by various contributors; 1929. Bristol: John Wright and Sons, Limited. Demy 8vo., pp. 610, with illustrations. Price: 20s. net.

THE MATRIX OF THE MIND, by Frederic Wood Jones, F.R.S., and Stanley D. Porteus; 1929. London: Edward Arnold and Company. Demy 8vo., pp. 432, with illustrations. Price: 21s. net.

Diary for the Month.

JUNE 18.—Tasmanian Branch, B.M.A.: Council.
JUNE 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 18.—New South Wales Branch, B.M.A. Organization and Science Committee.
JUNE 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 19.—Western Australian Branch, B.M.A.: Branch.
JUNE 19.—South Sydney Medical Association, New South Wales.
JUNE 19.—Section of Obstetrics and Gynæcology, New South Wales Branch, B.M.A.
JUNE 25.—New South Wales Branch, B.M.A.: Council (Quarterly).
JUNE 26.—Victorian Branch, B.M.A.: Council.
JUNE 27.—New South Wales Branch, B.M.A.: Branch.
JUNE 27.—South Australian Branch, B.M.A.: Branch.
JUNE 28.—Queensland Branch, B.M.A.: Council.
JULY 2.—Tasmanian Branch, B.M.A.: Council.
JULY 2.—Eye, Ear, Nose and Throat Section, South Australian Branch, B.M.A.
JULY 3.—Victorian Branch, B.M.A.: Branch.
JULY 3.—Western Australian Branch, B.M.A.: Council.

Medical Appointments.

Dr. C. H. Kellaway (B.M.A.), the Director of the Walter and Eliza Hall Institute for Research in Pathology and Medicine, has been elected a Fellow of the Royal College of Physicians of London.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

GYMPIE HOSPITAL BOARD: Medical Officer.
QUEEN VICTORIA MEMORIAL HOSPITAL FOR WOMEN AND CHILDREN, MELBOURNE: Junior Resident Medical Officer (Female).
SYDNEY HOSPITAL, SYDNEY: Honorary Relieving Assistant Physician.
ST. MARGARET'S HOSPITAL, SYDNEY: Resident House Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital. Toowoomba Friendly Societies Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are **£2 for Australia and £2 5s. abroad *per annum* payable in advance.**